

EATING DISORDERS AND THE REGULATION OF EMOTION: FUNCTIONAL
MODELS FOR ANOREXIA AND BULIMIA NERVOSA

by

DANYALE P. McCURDY

Submitted to the graduate degree program in Clinical Psychology
and the Graduate Faculty of the University of Kansas
in partial fulfillment of the requirements for the degree of
Doctor of Philosophy.

Nancy A. Hamilton, Ph.D.
Chairperson

Raymond L. Higgins, Ph.D.

Rick E. Ingram, Ph.D.

Alice Lieberman, Ph.D.

Kristopher Preacher, Ph.D.

Date defended: June 14, 2010

The Dissertation Committee for Danyale P. McCurdy certifies
that this is the approved version of the following dissertation:

EATING DISORDERS AND THE REGULATION OF EMOTION: FUNCTIONAL
MODELS FOR ANOREXIA AND BULIMIA NERVOSA

Nancy A. Hamilton, Ph.D.
Chairperson

Date approved: June 28, 2010

ABSTRACT

EATING DISORDERS AND THE REGULATION OF EMOTION: FUNCTIONAL MODELS FOR ANOREXIA AND BULIMIA NERVOSA

Danyale P. McCurdy

Department of Psychology

Doctor of Philosophy

Different types of eating disorders may be better described and understood in terms of their specific behaviors and the emotion regulatory function these behaviors serve. Individuals may influence their affective states by upregulating or downregulating different emotions. Evidence characterizing eating disordered behavior according to this theory is discussed based on personality research, comorbidity, affect intensity, and neurobiology. An original emotion regulation theory of eating disorders is proposed. This theory centers on individuals' affect intensity and their emotion regulation strategies. Eating disorders are conceptualized by their behavioral components, not by their diagnostic category. Individuals with anorexia nervosa-restricting type (restrictors) were compared to individuals with bulimia nervosa and anorexia nervosa-binge/purge type (binge-purgers). Restrictors were posited to be low in affect intensity, or emotionally constricted. In contrast, binge-purgers were posited to be high in affect intensity, or emotionally labile. Food restriction in restrictors was hypothesized to be a method for increasing positive affect and decreasing negative affect. In binge-purgers, bingeing was seen as a method for reducing negative affect, and purging was seen as a means to increase positive affect and reduce negative

affect. Participants were 63 inpatient females with a clinical diagnosis of an eating disorder. Participants were given an assessment battery measuring various indices of eating behavior and emotionality. Overarchingly, it was hypothesized that women classified as restrictors versus binge-purgers would show different patterns of emotional processing. Results of the present study support the theory that affective differences exist between individuals who solely restrict dietary intake and those who also engage in binge-purge behaviors. It appears that affect intensity may be one of the most important differences. Binge-purgers had marginally higher levels of affect intensity than did restrictors. However, affect intensity moderated the emotional outcomes of disordered eating behaviors in both groups. These preliminary analyses support the emotion regulation theory of eating disorders and warrant further investigation.

ACKNOWLEDGMENTS

I wish to express sincere appreciation to Professor Nancy Hamilton for her assistance in the preparation of this manuscript. This dissertation required many hours of her editing, patience, and moral support. I am forever grateful to have had the opportunity to work with her. She has always been an advocate for me and a model mentor that I aspire to resemble. I would also like to thank the rest of my committee for their generosity of time and valuable intellectual contributions: Professors Raymond Higgins, Rick Ingram, Alice Lieberman, and Kristopher Preacher. In addition, I would like to thank our team of research assistants in the Hamilton Lab. Special thanks goes to my colleague and friend, Natalie Stevens, for her time and assistance over the past year. She has gone above and beyond by helping organize and facilitate data collection and entry. Finally, I would like to add a very special and sincere thank you to Dr. Hamilton, Dr. Higgins, and Dr. Cary Savage for their continued guidance and support throughout all of my endeavors, past, present, and future. You all have influenced me more than you will ever know.

Dedicated to my mother and father. With their unwavering love, support,
and patience, they enabled the first college graduate in our family to
become the first doctoral graduate.

TABLE OF CONTENTS

CHAPTER I: INTRODUCTION AND LITERATURE REVIEW.....	1
Anorexia Nervosa and Bulimia Nervosa: Diagnostic Features.....	2
Eating Disorders and Affective Comorbidity.....	4
Personality and Temperament Characteristics Associated with AN and BN.....	7
Summary of Personality Literature.....	9
Theoretical Model: Affect Intensity and Emotion Regulation.....	10
Affect Intensity.....	10
Emotion Regulation.....	11
Affect and Emotion Regulation in Eating Disorders.....	13
Model for Restrictors.....	17
A Broader Context.....	17
Neurobiological Evidence.....	19
Restrictor Model Summary.....	21
Model for Binge-Purgers.....	22
A Broader Context.....	22
Neurobiological Evidence.....	26
Binge-Purger Model Summary.....	28
Summary.....	29
Purpose of the Present Study.....	30
Hypothesis 1: Behavioral versus Diagnostic Profiles.....	30

Hypothesis 2: Affective Constructs in Restrictors and Binge-Purgers.....	30
Hypothesis 3: Restricting Behavior and Affective Outcomes.....	31
Hypothesis 4: Binge-Purge Behavior and Affective Outcomes.....	32
CHAPTER II: METHOD.....	33
Participants.....	33
Measures.....	33
Diagnostic Classification.....	33
Affect Intensity.....	37
Affect.....	37
Emotion Regulation.....	38
Symptoms of Depression.....	39
Procedure.....	39
Data Analysis.....	41
CHAPTER III: RESULTS.....	42
Power Analysis.....	42
Hypothesis 1: Behavioral versus Diagnostic Profiles.....	42
Profile Comparison.....	42
Affect Intensity.....	43
Depressive Symptomatology.....	43
Weekly Negative Affect.....	44
Weekly Positive Affect.....	44

Hypothesis 2: Affective Constructs in Restrictors and Binge-Purgers.....	44
Tonic Differences.....	44
Phasic Differences.....	45
Hypothesis 3: Restricting Behavior and Affective Outcomes.....	45
Correlational Analyses for Restrictors.....	45
Emotion Regulation and Response to Restricting.....	46
Hypothesis 4: Binge-Purge Behavior and Affective Outcomes.....	48
Correlational Analyses for Binge-Purgers.....	49
Emotion Regulation and Response to Restricting, Binging, and Purging.....	49
CHAPTER IV: DISCUSSION.....	54
Summary of Findings.....	54
Differences between Restrictors and Binge-Purgers.....	54
Within Group Differences.....	55
Restrictors.....	55
Binge-Purgers.....	57
Future Directions.....	59
Treatment Implications.....	62
Limitations.....	64
Conclusions.....	66
REFERENCES.....	67
TABLES.....	81

FIGURES.....	87
APPENDICES.....	94
A. Eating Disorder Diagnostic Scale.....	94
B. Affect Intensity Measure.....	95
C. Positive and Negative Affect Schedule – Modified to Assess Eating Behaviors.....	97
D. The Emotion Amplification and Reduction Scales.....	101
E. Informed Statement of Consent.....	102
F. Recruitment Letter and Instructions.....	105
G. Demographic Questionnaire.....	106
H. Power Analysis.....	107

LIST OF TABLES

Table 1: Descriptive Statistics.....	81
Table 2: Correlation Matrix.....	82
Table 3: ANCOVA: Behavioral versus Diagnostic Profiles.....	83
Table 4: Independent Samples T-test: Restrictors versus Binge-Purgers.....	84
Table 5: Restrictor Correlation Matrix.....	85
Table 6: Binge-Purger Correlation Matrix.....	86

LIST OF FIGURES

Figure 1: Behavioral versus Diagnostic Profiles.....	87
Figure 2: Restrictors versus Binge-Purgers.....	88
Figure 3: Correlational Prediction for Restrictors.....	89
Figure 4: Correlational Predictions for Binge-Purgers.....	90
Figure 5: Restrictors Interaction: Emotion Reduction x Affect Intensity.....	91
Figure 6: Binge-Purgers Interaction: Emotion Amplification x Affect Intensity.....	92
Figure 7: Binge-Purgers Interaction: Emotion Reduction x Affect Intensity.....	93

CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

Eating disorders are some of the most chronic and difficult to treat of all psychological illnesses. Approximately 0.5–1.0% of late adolescent or adult women meet criteria for the diagnosis of anorexia nervosa (AN), and approximately 1–2% meet diagnostic criteria for bulimia nervosa (BN) (APA, 2000). Course and treatment outcomes for AN reveal that approximately one-half will recover, 10% will remain chronically ill, and 10% will die as a consequence of their illness (Strober, Freeman, & Morrell, 1997; Sullivan, 1995). Similarly for BN, approximately one-half will recover, one-third will continue to have some symptoms of the disorder or relapse, and approximately 20–30% will remain chronically ill (Keel & Herzog, 2004). The treatment refractory nature of eating disorders has led some theorists to suggest that there may be some functional value of AN and BN, and their associated behaviors. Food restriction and binge-purge behaviors may serve an emotion regulatory function in these individuals. Examining the possible relationship between emotion regulation and disordered eating behavior may help researchers and clinicians develop more effective interventions for disorders that historically have been very difficult to treat.

Although first described in the psychological literature a century ago, clinicians and researchers are still struggling to understand the etiology, neurobiology, and treatment of eating disorders. The difficulties inherent in treating eating disorders, as well as their high comorbidity with mood disorders, have led researchers to investigate the shared psychobiological underpinnings of affect and

eating behavior. Furthermore, the unique differences between individuals who solely restrict their food intake and those that also engage in binge-purge behaviors may be indicative of affective differences between subtypes of eating disorders. More specifically, particular eating disorder behaviors may be serving different emotion regulatory functions.

The present study aims to present a theoretical perspective examining the relationship between specific eating disordered behaviors and the emotion regulatory functions they serve. Current thinking in the emotion regulation literature holds that emotions can be regulated by increasing or decreasing the magnitude of a given emotion (Gross, 1998a, 1998b, 2001; Hamilton et al., 2009). The current study proposes that different subtypes of eating disorders can be better described and understood according to this model. Evidence for this theory will be discussed from research on personality, comorbidity, affect intensity, and neurobiology.

Anorexia Nervosa and Bulimia Nervosa: Diagnostic Features

The *Diagnostic and Statistical Manual of Mental Disorders/Text Revision (4th ed.) (DSM-IV)* (APA, 2000) defines eating disorders in terms of behaviors, but also categorically differentiates diagnoses based on body weight. BN is characterized by frequent episodes of binge eating and recurring inappropriate compensatory behaviors in order to prevent weight gain. Criteria stipulate that binge eating and compensatory behaviors must occur at least twice a week for at least three months. In addition, self-image is typically tied to body shape and weight (APA, 2000). Binges are characterized by the consumption of a large amount of food within a discrete period

of time, with a sense of lack of control over eating during the episode. Compensatory behaviors are defined as actions intended to prevent weight gain. These behaviors could include: vomiting; use of laxatives, diuretics, and/or enemas; fasting; and/or excessive exercise. In addition to these behavioral criteria, one of the diagnostic criteria for BN is a preservation of normal body weight.

In contrast, AN is characterized by the *DSM-IV* primarily as a refusal to maintain a normal body weight (less than 85% ideal weight). Typically, in clinical settings, this cut-off is somewhat arbitrarily set at a body mass index of 17 or below. AN is also characterized by: an intense fear of gaining weight even though underweight, a disturbance in body or shape perception, and amenorrhea. The disorder manifests as either a restricting type (AN-R) or a binge/purge type (AN-BP). The binge/purge subtype is distinguished from the restricting subtype by the presence of bingeing and purging (as described in BN). Individuals with the restricting subtype exclusively restrict their caloric intake and also may exercise, although not for purgative purposes. So, the sole factor differentiating BN from AN-BP is percentage of ideal weight.

The *DSM-IV* clearly appears to distinguish eating disorders more in terms of body weight than in terms of eating behaviors. The criteria for BN and AN-BP overlap in terms of behaviors, and the only differentiating factor is body weight. Therefore, a patient could be diagnosed with AN-BP initially, and following weight restoration could be diagnosed with BN. This causes confusion both clinically and in research. Although the *DSM-IV* distinguishes the two subtypes of AN from BN,

differentiating eating disorders in terms of behavior (i.e., restricting versus binge-purging) warrants further examination. Focusing on behaviors, instead of a metabolic outcome, offers the real possibility of understanding the functional significance of these behaviors, particularly as they relate to emotion regulation.

Eating Disorders and Affective Comorbidity

Although the relationship between eating disordered behavior and emotion regulation has not yet been well established, there is a wealth of literature documenting eating disorders' comorbidity with mood disorders and Cluster B personality disorders. Depression is the most commonly diagnosed comorbid disorder (O'Brien & Vincent, 2003). The lifetime prevalence rate of major depression among individuals with AN is between 46% and 74% (Casper, 1998). Among those with AN-R, 15–50% have major depression at some point in their lives, and among those with AN-BP, the lifetime prevalence rate of depression is even higher, ranging from 46–80% (Casper, 1998). The lifetime prevalence rate of major depression in BN is 50–65% (Casper, 1998). The rates of depression among those with eating disorders can be compared to the much lower lifetime prevalence rate (17%) of major depression in the general population (APA, 2000). Conversely, women with a diagnosis of major depression have higher lifetime prevalence rates of eating disorders. Approximately 1–7% of women with major depression will develop AN in their lifetime and approximately 9–21% of these individuals will develop BN in their lifetime (Carter, Joyce, Mulder, Luty, & Sullivan, 1999; Fava et al., 1997). Furthermore, the presence of depression among those with eating disorders is

associated with higher rates of substance abuse and poorer treatment outcomes. The high rates of depression among those with AN-BP and BN suggests an important relationship between bingeing/purging and mood.

Suicidality offers another indication of emotion dysregulation. Individuals with eating disorders have significantly higher rates of suicide attempts and completed suicides than the rest of the population, which may be related to mood disturbance. AN has the highest mortality rate of any psychiatric disorder (Sullivan, 2002) and among AN patients, suicide is the second leading cause of death (after death from medical complications). Among those with AN, 2.5% die from suicide, and severity of depression has been found to be a predictor of suicide in this population (Franko & Keel, 2006). While completed suicide is higher among those with AN compared to those with BN, the presence of bingeing-purging behavior significantly increases the risk of suicide attempts which occur in up to 20% of patients with AN, and in up to 35% of patients with BN (Franko & Keel, 2006). Higher rates of depression have been found among BN attempters than AN attempters; however, severity of depression does not prospectively predict suicidality in BN. The high rate of suicide attempts within these disorders provides more evidence that eating disorders are related to emotion dysregulation and also underscores the need for a greater understanding of the role of affect regulation. Moreover, the epidemiological data suggest that it may be useful to examine depression and suicidality in relation to bingeing, purging, and restricting behaviors, as

opposed to *DSM-IV* diagnostic groups, which are primarily based on metabolic outcomes.

Examining the high rates of personality pathology among those with eating disorders provides further evidence of a relationship between eating pathology and emotion regulation. A meta-analysis revealed different levels of comorbidity between types of eating disorders and types of Axis II pathology (Rosenvinge, Martinussen, & Ostensen, 2000). Higher rates of Cluster B (antisocial, borderline, histrionic, and narcissistic) personality disorders were found in BN (44%) compared to AN (15%), with borderline personality disorder (BPD) occurring most frequently (31% in BN compared to 14% in AN). Higher rates of Cluster A (paranoid, schizoid, and schizotypal) personality disorders were also found in BN (27%) compared to AN (12%). However, similar rates of Cluster C (avoidant, dependent, and obsessive-compulsive) personality disorders were found in AN and BN (~45%). Unfortunately, this study did not distinguish between AN-R and AN-BP.

BPD is among the most discussed of comorbid Axis II pathologies in eating disorders. Interestingly, the hallmark of BPD is intense emotion dysregulation (APA, 2000). A number of studies have found higher rates of BPD in individuals that engage in bingeing and purging, both BN and AN-BP (e.g., Carroll, Touyz, & Beumont, 1996; Herzog et al., 1992). Moreover, higher rates of eating pathology have been observed among individuals with BPD. In a large study of psychiatric inpatients (N = 504), 62% of females with BPD also met criteria for an eating disorder (Zanarini et al., 1998). Based on the comorbidity of BPD with eating disorders, and their shared

emotion regulation difficulties, this relationship warrants further exploration.

Furthermore, differential personality pathology findings within AN and BN may be of interest when distinguishing and characterizing subtypes of eating disorders.

Personality and Temperament Characteristics Associated with AN and BN

Research has suggested that AN can be differentiated from BN in terms of personality characteristics. Personality differences have been documented across multiple studies and using a variety of assessment methodologies, ranging from expert clinical evaluation to standard personality batteries. More importantly, these outcomes have also pointed to behavioral distinctions. Specifically, binge-purge behaviors and restricting behaviors are related to distinct personality profiles.

For instance, clinicians reliably discriminate individuals with AN from individuals with BN based on characteristics of emotional processing (Westen & Harnden-Fischer, 2001). A large number (176) of experienced clinicians (including psychologists and psychiatrists) were asked to describe a patient who met full *DSM-IV* criteria for AN or BN. The clinicians completed a demographic questionnaire about their patient, a 1–7 rating of the degree that the patient met criteria for each of the *DSM-III-R* and *DSM-IV* personality disorders, and a Q-sort procedure that assessed personality features and affect regulation. Raters identified three subgroups of patients: a constricted/overcontrolled group, an emotionally dysregulated/undercontrolled group, and a high-functioning/perfectionistic group. The first two subgroups are of particular interest for the topic at hand. The constricted/overcontrolled group had the largest number of AN-R (35%) in the study. These

individuals were found to constrict or restrict many aspects of their life and they tended to feel depressed, anxious, ashamed, and anhedonic. The emotionally dysregulated/undercontrolled group was made up of 50% AN-BP and 50% BN. These individuals tended to experience intense, poorly regulated emotions, and desperately sought relationships in an effort to soothe emotional distress.

In an earlier review of studies on temperament and personality pathology among eating disordered individuals, researchers (Vitousek & Manke, 1994) suggested that temperament and personality pathology can help identify meaningful distinctions among women with different eating disorder profiles. When results from a literature review were analyzed, individuals with AN-R had the most consistent profile overall. They were found to be reticent, constricted, and conforming children, and to display profound obsessional personality features during the active eating disorder phase. Lower weight individuals with BN and those with AN-BP were found to be high in negative affect and appeared slightly more impaired than patients with other profiles. Although the individuals that binged and purged shared some traits with AN-R, they were identified as being more outgoing, affectively labile, and difficult as children.

The studies of personality pathology among those with eating disorders (Westen & Harnden-Fischer, 2001; Vitousek & Manke, 1994) identified distinguishing characteristics between eating disordered individuals who solely restricted their food intake and those who also binged and purged. To summarize, individuals with AN-R were described as overcontrolled, anhedonic, and constricted.

In contrast, those who engaged in bingeing and purging behavior (both BN and AN-BP) appeared to be more undercontrolled, volatile, and emotionally labile. So, personality profiles were not as closely related to diagnostic categorization (i.e., AN, BN) as behavioral profiles. Specifically, women who binged and purged were classified differently from those who exclusively restricted food intake. Thus, it may be more useful to characterize individuals with eating disorders in terms of their eating behavior than in terms of their diagnosis alone.

Consistent with this formulation, some researchers have reviewed studies reporting taxometric analyses of eating disorders (Williamson, Gleaves, & Stewart, 2005). They reported that disorders involving binge eating (i.e., BN, AN-BP, and binge eating disorder) lie on a separate continuum from AN-R. Restricting behaviors could be described on a single unique continuum, regardless of whether they occurred in individuals with AN, non-eating-disordered normal weight individuals, or those who were obese. This suggests that eating disorders may be better conceptualized dimensionally, and by the underlying taxons of bingeing and restricting.

Summary of Personality Literature. The links between eating disorders and Axis I and II pathology are informative, and suggest that dimensions of emotional processing may play a role in maintaining eating disordered behavior. The personality literature supports the hypothesis that individuals who exclusively restrict food intake may differ from those who binge and purge in terms of their emotional lability, or affect intensity, and their ability to manage or control emotional responses (Vitousek & Manke, 1994; Westen & Harnden-Fischer, 2001). Furthermore, based on the high

comorbidity with mood and personality disorders, it seems likely that eating behaviors may serve to regulate both negative and positive affect. Unfortunately, the aforementioned studies do not provide specific information about the nature of the shared disorders. Offering further insight and complementing the psychiatric literature are studies that directly examine the role of emotional processing in patients with eating disorders.

Theoretical Model: Affect Intensity and Emotion Regulation

Emotional processing can be conceptualized in terms of arousal and control (Derryberry & Rothbart, 1988). Emotional arousal is likely to be related to autonomic arousal systems (Eysenck, 1981) and may explain dimensions of personality (e.g., introversion and extroversion). Reactivity levels within cortical pathways may influence one's optimal level of arousal and stimulation. For example, individuals who are more reactive may be more neurotic in nature and show stronger and more variable emotional reactions (Derryberry & Rothbart, 1988). Emotional control, on the other hand, is related more to self-regulation and effortful control of attention to stimuli. Shifting attention toward positive stimuli may serve to enhance or preserve arousal and emotion; whereas, shifting attention away from negative stimuli may serve to attenuate or restrain arousal and emotion (Derryberry & Rothbart, 1988). This premise of autonomic arousal and effortful control may help us better understand affect intensity and emotion regulation among those with eating disorders.

Affect Intensity. One way in which autonomic arousal can be conceptualized is in terms of affect intensity. Affect intensity refers to the strength with which

individuals experience their emotions (Larsen & Diener, 1987). This construct is defined as a stable trait that generalizes to both positive and negative emotions. There are distinguishing characteristics between individuals who are high and those who are low in affect intensity. Individuals high in affect intensity are theorized to react more intensely to daily life events (Larsen, Diener, & Emmons, 1986) and tend to have distinct patterns of event-related cognitions (e.g., increased focus on emotional content and personal relevance of the event) (Larsen & Diener, 1984). People with high affect intensity show quicker and more frequent shifts in their mood throughout the day and experience more variable emotional lives in general. Larsen and Diener (1987) describe individuals high in affect intensity as having an emotional life distinguished by its abruptness, changeableness, and volatility.

Individuals with low affect intensity are known to restrict emotional stimulation (Larsen & Diener, 1984, 1987) and, in contrast to those high in affect intensity, are theorized to have less frequent shifts in daily mood shifts and to be less active, less sociable, and less physically arousable. Additionally, people who are low in affect intensity are theorized to have emotional lives characterized by enduringness, consistency, and stability. The dimension of intensity seems critically important given the wealth of literature documenting differences between individuals with AN and BN in terms of basic dimensions of temperament.

Emotion Regulation. In addition to dispositional differences in affect intensity, individuals also control or regulate their emotions in different ways. Current thinking in the emotion regulation literature supports the view that individuals employ

different strategies at different time points in order to upregulate or downregulate their emotions (Gross, 1998a, 1998b, 2001). Gross describes a process model of emotion regulation wherein he posits that an emotional response unfolds over a distinct period of time. At different points along this emotional timeline, one can employ different strategies to influence emotion. The emotional timeline can be broken down into response-focused and antecedent-focused emotion regulation strategies (Gross, 1998b). So, people are able to manipulate their emotional state via a number of strategies. For instance, one might choose to prolong or intensify an existing emotion, or reduce the magnitude and/or duration of an existing emotion.

Although Gross and others have argued that emotion regulation should be described in terms of regulating both positive and negative emotions, most existing measures of emotion regulation define emotion regulation exclusively in terms of downregulating negative emotions. In contrast, The Emotion Amplification and Reduction Scales (TEARS) separately measure upregulation and downregulation of emotion in general (Hamilton et al., 2009). As would be expected, increasing and decreasing emotions were found to have different emotional correlates. Specifically, individuals who reported being able to limit or reduce an emotional state by selecting an emotional response, or alter an emotional state by softening, shortening, or stopping it, also reported fewer symptoms of depression, lower fatigue, and less negative affect. Interestingly, those who were better able to intensify a preexisting emotional state had higher levels of both positive and negative affect, as well as

greater fatigue (Hamilton et al., 2009). Thus, depending on one's specific emotion regulatory skills, there may be a direct and cumulative effect on affective states.

Affect and Emotion Regulation in Eating Disorders

Distinguishing among strategies for increasing or decreasing emotions may be critically important in the context of eating disorders. Among those with an eating disorder, behaviors such as restricting and binge/purging serve as regulatory strategies, thus serving to reinforce the behavior. It has been theorized that eating disordered individuals have deficits in emotion processing related to an impaired ability to identify their emotions and have a lower level of emotional awareness in general (Bydlowski et al., 2005). Various studies support the presence of these emotional deficits in eating disorders as well as distinctions between those who solely restrict and those who binge and purge. Individuals with AN-R have been found to have significantly more difficulties in both emotion recognition and regulation compared to healthy controls (Harrison, Sullivan, Tchanturia, & Treasure, 2009). A recent functional magnetic resonance imaging (fMRI) study showed that individuals with BN and AN-BP had greater medial prefrontal cortex (mPFC) activation when processing emotionally negative words related to their bodies compared to AN-R and control subjects (Miyake et al., 2010). The mPFC is known for its role in emotional processing. Based on both measures of self-report and neurobiological data, emotional difficulties are likely inherent in eating disorders.

Data suggest that problems with emotional processing are present early on in individuals with disordered eating. Among adolescents girls (ages 10-15), those with

high negative affect, difficulties identifying emotions, and high levels of maladaptive coping strategies were significantly more likely to report symptoms of disordered eating (Sim & Zeman, 2006). So, difficulty regulating negative affective states was directly related to dysfunctional eating behaviors among young girls. Studies such as this highlight the importance of understanding the affective vulnerabilities to disordered eating among young girls.

Accordingly, individuals with different types of eating disorders have been shown to exhibit differing attachment styles and to cope differently with emotional situations. Responses to relationships and emotional arousal differ between those who restrict and those who binge and purge. In AN-R, the dominant attachment style is fearful/avoidant; whereas, in binge-purge disorders, it is anxious but relatively more secure (Turner, Bryant-Waugh, & Peveler, 2009). Individuals with AN-R have been found to use passive coping styles, while individuals with AN-BP or BN have been found to use more active coping styles (Turner et al., 2009). Passive coping in AN-R includes dependence upon others for approval and nurturing and may include dietary restriction. Alternatively, active coping in AN-BP and BN is more problem-focused and may comprise bingeing and vomiting. This type of emotional processing distinction may have clinical utility and highlights differences between binge-purge and restricting disorders in terms of emotional processing.

Although not focusing on emotion regulation specifically, other influential models of eating disorders have emphasized that emotional dysregulation may lead to disordered eating patterns. Stice and colleagues proposed a dual pathway model for

BN that is consistent with this premise (Stice, Nemeroff, & Shaw, 1996). Their model suggests that restrained eating is a response to negative affect and that body dissatisfaction also increases negative affect, which contributes to bulimic symptomatology (binging and purging). This model clearly suggests ties between various emotions and eating disorder behavior.

Eating disorders have also been linked to a number of different negative affective states. In particular, eating disorders and their associated behaviors have been linked to anger (Waller et al., 2003). Women with eating disorders were found to have higher levels of state anger and to suppress their anger more than women without eating pathology. This was especially true for women who binged and purged. The authors theorized that differences in purging measures (vomiting, exercise, or laxative abuse) reflected different emotional functions. Women who binged and vomited reported higher levels of trait anger; excessive exercisers reported higher levels of state anger; and laxative abuse was associated with anger suppression. These findings further support the notion that different eating disordered behaviors may serve varied purposes in terms of emotionality.

Interestingly, various studies have pointed to a connection between eating behavior and emotion dysregulation in non-clinical populations. It is generally recognized that eating behaviors are affected by and associated with emotions (Desmet & Schifferson, 2008). Emotion regulatory eating is defined as goal-oriented behavior designed to decrease an unpleasant feeling state (Booth, 1994). An example of emotion regulatory eating could involve eating more of a satisfying food in order

to decrease a negative affective state. For instance, in a study looking at ice-cream consumption, women who scored high on an emotional eating scale (meaning they were more likely to respond to negative emotional stimuli by eating) consumed more ice-cream under controlled conditions than women who scored low on emotional eating (van Strien, 2000). This study suggests that the link between negative affect and bingeing is not unique to those with clinically significant levels of eating disorder symptomatology.

Although eating disorder behaviors are thought to have a functional, emotion-regulatory value, these behaviors do not develop or occur in a cultural vacuum. The sociocultural model of eating pathology hypothesizes that social pressure to be thin fosters an internalization of the thin ideal and body dissatisfaction, placing individuals at risk for dieting, negative affect, and eating pathology (Stice, 2002; Striegel-Moore, Silberstein, & Roden, 1986). Internalizing the thin-ideal and placing too much value on the importance of appearance promotes body dissatisfaction which, in turn, promotes dieting, negative affect, and eating pathology (Garner, Olmstead, & Polivy, 1983; Stice, 2002). In other words, societal pressures are related to vulnerability to eating disorders and also to maintenance of symptoms.

Although these models suggest that societal pressures and internalization of the thin ideal relate to all eating disorders, different vulnerability factors (such as affect intensity) and different maintenance factors (such as emotion regulation), are likely to influence the specific behaviors exhibited (i.e., dietary restriction versus bingeing-purging). Understanding the affective differences between those who solely

restrict and those who binge-purge may facilitate a better understanding of the functional value of these dysfunctional behaviors.

Model for Restrictors

The following section describes a model hypothesizing that restricting behaviors in AN-R serve an emotion regulatory function. As posited in the theoretical model, individuals who solely restrict their food intake are emotionally constricted and are likely to be low in affect intensity. Within the proposed model, restricting behaviors are posited to help regulate emotion by facilitating positive affect and reducing negative affect.

A Broader Context. A number of risk and maintenance factors have been explored in individuals with eating disorders. For individuals who restrict, body dissatisfaction is hypothesized to be associated with dieting (based on the belief that this is an effective weight control technique) and negative affect (because appearance is an essential criterion for self-evaluation), which then increase the risk of eating pathology (i.e., food restriction) (Crisp, 1984; Stice, 2002). Body dissatisfaction is one of the most consistent and robust risk and maintenance factors for disordered eating behaviors (Stice, 2002).

Interestingly, such restricting behaviors as limiting food intake have been found to increase positive affect. Losing weight and successfully avoiding food serve a reinforcing and motivational purpose (Vitousek & Ewald, 1993). Sensations of euphoria related to feelings of success, superiority, control, and attention positively reinforce restricting behavior in AN. Research also has shown correlations between

positive emotions and disordered eating attitudes, disordered eating behaviors, and psychological themes among eating disordered individuals (Overton, Selway, Strongman, & Houston, 2005). Moderately strong positive correlations were found between positive emotions as measured by *The Differential Emotions Scale-IV* (DES-IV; Blumberg & Izard, 1985) and *Eating Disorder Inventory-2* (EDI-2; Garner, 1991) subscales (drive for thinness, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness, maturity fears, asceticism, impulse regulation, social insecurity). Cumulatively, these data suggest that restricting behaviors may be purposefully used to upregulate positive affect.

Although affect intensity has not been examined in relation to eating disorders, many of the personality characteristics of people with AN are consistent with low affect intensity. Individuals with AN have intolerance for ambiguity and are quite uncomfortable with both the feeling and expression of strong affect due to its unpredictable nature (Vitousek & Ewald, 1993). Women with AN have also been found to have a lower level of emotional awareness than BN individuals (Bydlowski et al., 2005). Consistent with this theory, AN-R has been linked with alexithymia, a personality style defined by difficulty identifying, describing, and differentiating emotions (Sexton, Sunday, Hurt, & Halmi, 1998). Researchers have also documented that the personality traits of perfectionism and harm avoidance (Cockell et al., 2002) may be risk factors for eating pathology. Perfectionism also tends to persist, even after recovery, in women with AN (Santonastaso, Friederici, & Favaro, 1999). Additionally, intense feelings in harm avoidant (characterized by anxiety, pessimism,

fear, and doubt) individuals may be intolerable based on their discomfort with strong emotions (Kaye, Frank, Bailer, & Henry, 2005). Body dissatisfaction, trait perfectionism, and harm avoidance all appear to play a role in the development and maintenance of this disorder. They may also be related to mood disturbance in AN. With all of this taken into account, it can be hypothesized that restrictors are likely to be low in affect intensity.

Neurobiological Evidence. This model suggests an important upstream role for personality and sociocultural factors; however, emotion regulation may play a strong supporting role in maintaining dysfunctional behaviors. Brain serotonin (5-HT) systems are known to modulate appetitive behaviors (Blundell, 1984) as well as mood. Accordingly, two commonly found traits in this disorder, harm avoidance and perfectionism, are associated with alterations in brain 5-HT function among those with AN (Kaye et al., 2005; Steiger, 2004). Along with these temperament features, emotion regulation may play a powerful supporting role in maintaining anorectic symptoms.

Biological data on AN have identified 5-HT as a substantial maintenance factor. Kaye et al. (2003) proposed an influential model suggesting that individuals with AN are hyperserotonergic compared to non-eating-disordered individuals, and use food restriction as a means of reducing levels of 5-HT. Initially, increased levels of 5-HT may cause food to be less pleasurable and allow for rapidly developing satiety in AN. However, dietary restriction would produce a reduction of tryptophan (the precursor of 5-HT). Corresponding with this model, during the active phase of

illness, individuals with AN have a significant reduction in cerebrospinal fluid concentrations of 5-HT metabolites (e.g., 5-hydroxyindoleacetic acid [5-HIAA]). Also consistent with the 5-HT model, after recovery, individuals with AN had much higher than normal concentrations of these metabolites (Kaye et al., 2005). Importantly, research has shown that in this population, high levels of 5-HT are associated with increased levels of anxiety and dysphoric mood (Kaye et al., 2003). Restricting food intake may serve the functional purpose of decreasing anxiety and dysphoria. This finding is consistent with the clinical observation that many anorectics prefer a vegetarian lifestyle and often have a strong distaste for red meats and dairy (which possess high levels of tryptophan).

The above data are interesting, although the direction of the relationship is questionable. However, experimental data are consistent with the 5-HT regulation hypothesis. Currently ill individuals with AN were compared to individuals recovered from AN and normal controls following an acute tryptophan depletion challenge (Kaye et al., 2003). At baseline, both ill and recovered women with AN had higher blood plasma levels of tryptophan compared to control subjects. When participants were deprived of tryptophan, both ill and recovered AN reported a significant reduction in anxiety, whereas no significant difference in anxiety symptomatology was found among controls. This study offers very compelling evidence for the hypothesis that starvation-induced reduction of 5-HT may serve as an emotion regulatory strategy that is specific to individuals with AN.

Brain imaging studies using positron emission tomography have found a distinction between AN-R and AN-BP as well. Compared to AN-BP, recovered AN-R had reduced 5-HT_{2A} receptor activity in the pregenual cingulate cortex (Frank et al., 2002). This area has been tied to a number of emotional operations including assessing the salience of emotional and motivational information and the regulation of emotions (Bush, Luu, & Posner, 2000). These neuroimaging data lend further support to the emotion regulatory function of dietary restriction and also support the assertion that eating disorders should be distinguished more in terms of specific eating disordered behavior. In this population, dietary restriction may be affectively adaptive because restriction modulates mood at both the psychological and physiological level.

Restrictor Model Summary. In sum, this model posits that restrictors limit their food intake for both psychological and physiological rewards. Restriction of food serves as a means to increase positive affect and decrease negative affect. The latter emotion regulatory goal is likely to be mediated via reductions in 5-HT, which would have the effect of curbing anxiety and dysphoria. These affective states are likely to be extremely unpleasant for individuals who are generally posited to be low in affect intensity. The restriction of food and subsequent mood regulation may then be entangled in a positive feedback loop, where each piece reinforces the other repeatedly in a cyclic manner. The emotionally reinforcing consequence of food restriction may account for the chronicity and treatment refractory nature of this disorder.

Model for Binge-Purgers

The following section describes a model hypothesizing that bingeing and purging behaviors, in both BN and AN-BP, serve an emotion regulatory function. As posited in the theoretical model, binge-purgers are emotionally labile and are likely to be high in affect intensity. Within the proposed model, restricting, bingeing, and purging behaviors are posited to help regulate emotion via a number of pathways that influence both positive and negative affect.

A Broader Context. Among binge-purgers, the sociocultural model of eating disorders proposes that body dissatisfaction promotes dieting, as well as negative affect, which then increases the risk for eating pathology (including compensatory behaviors like vomiting) (Crisp, 1984; Stice, 2002). Body dissatisfaction may also help maintain bulimic symptoms (Stice, 2002), because individuals who binge and purge do so because they believe purging will help control the potential weight gain (and the negative emotions) from bingeing. Research supports the idea that bingeing-purging behaviors are inextricably linked to affect and mood, and further, they may act as possible emotion modulators within this population.

Although similar to the sociocultural model, the affect regulation model posits that individuals binge eat to provide relief and distraction from unpleasant emotions (McCarthy, 1990; Stice, 2002). Consequently, many individuals engage in drastic compensatory behaviors to reduce anxiety about future weight gain. In other words, bingeing may be used as a means of reducing negative emotions. It has been reported that some individuals find compensatory behaviors like purging to be emotionally

cathartic (Hawkins & Clement, 1984). Thus, purging may simultaneously reduce negative affect and increase positive affect. Accordingly, negative affect is a causal risk factor for body dissatisfaction and eating pathology and a causal maintenance factor for binge eating among those with an eating disorder (Stice, 2002). Among binge-purgers, negative affect leaves one vulnerable to eating pathology, and binge-purge behaviors are reinforcing in that they serve to regulate both positive and negative affect.

Consistent with this model, studies have shown that when individuals diet or restrain their eating, they are left more vulnerable to overeating or bingeing. When restrained eaters are given a fattening preload (milkshakes, for example), they actually consume more food during subsequent portions of the experiment than if they were given no preload (Herman & Polivy, 1980, 1988; Herman, Polivy, & Esses, 1989). In other words, restrained eaters are vulnerable to the abstinence violation effect. The opposite is true for nondieters and unrestrained eaters. When unrestrained eaters receive a fattening preload, they consume less than if they were given no preload. Presumably, non-restrained eaters' behavior is guided by principles of energy regulation. Specifically, it would be expected that people would compensate for the preload by reducing subsequent food consumption. In contrast, once the fattening food interrupts the diet of a restrained eater, these individuals overindulge when more food is provided (Herman & Polivy, 1988). Importantly, when restraint is broken, individuals with eating pathology are likely to have an increase in negative

affective states (including anxiety, guilt, and shame) associated with gaining weight and the diet failure.

Both bingers and restrictors restrain eating, but one of the key differences between the two is that binge-purgers are higher in emotional lability (affect intensity). These individuals may be more easily disinhibited, have more negative affect to contend with in the first place, and have fewer resources to cope. BN has been linked with impaired emotional intelligence (Markey & Vander Wal, 2007). Emotional intelligence is a variable of emotion regulation, and lower levels are associated with a poorer ability to control emotions effectively and cope with challenges. Therefore, low levels of emotional intelligence in BN may be associated with greater emotion dysregulation.

Among binge-purgers, there are many mood related implications of the binge-purge cycle. Restraint theory suggests that when individuals restrict their dietary intake, they eventually have a decreased sensitivity to their own internal cues for hunger, and thus may rely on contextual cues to signal hunger and satiety (Herman & Mack, 1975). Within the proposed model, the cue of negative affect may encourage binge eating. Restrained eating is theorized to contribute to negative affect, and body dissatisfaction has been found to contribute to bingeing and purging by way of negative affect in individuals with BN (Stice et al., 1996). Following this logic, purging behaviors may act to repair negative affect. Data consistent with this formulation emerged from a more qualitative evaluation of the motivations to engage in bingeing and purging (Jeppson, Richards, Hardman, & MacGranley, 2003). Trained

clinicians interviewed women with BN (60-minute semi-structured interviews). Patients' responses were analyzed and five clusters of themes emerged; one of which was 'Attempts to regulate emotion.' Unpleasant emotion was more often than not a precursor to a large number of binge-purge episodes and the eating disorder behavior served as relief from the negative affective state. Binging and purging primarily served to minimize or repair unpleasant emotions (Jeppson et al., 2003). In other words, these individuals may binge and purge as a means to decrease negative affect and increase positive affect.

Another study examined mood and eating behavior in the natural environment using ecological momentary assessment (EMA) (Wegner et al., 2002). Women with subclinical binge eating behavior self-monitored their mood and eating behavior seven times per day over a two-week time period. The study found that binging occurred more often on days when mood was more negative (elevated anger, depression, and/or guilt/self-blame). However, binges were not predicted by momentary increases in negative mood and the binge, reportedly, did not increase positive affect nor decrease negative affect. In fact, when the participants self-initiated mood reports immediately after the binge, they reported increased levels of depressive mood and recalled that positive affect had been higher before the binge (Wegner et al., 2002). Taken together with other data, the EMA data suggest that binging may be motivated by high levels of negative affect; however, it is ultimately an unsuccessful way to repair negative mood. In fact, binging appears to leave the individual in a more dysregulated state, with greater negative affect, and less positive

affect. Hence, in the binge-purge population, subsequent purging may be a necessary strategy to reduce negative affect and increase positive affect.

Neurobiological Evidence. Clearly disposition and sociocultural factors have an important role in the development and maintenance of binge-purge symptomatology. As discussed, most etiological theories for binge-purgers include mood as a robust factor. The dual pathway model for BN (Stice et al., 1996) posits that restrained eating contributes to negative affect and that body dissatisfaction contributes to bulimic symptomatology (binging and purging) via negative affect. Negative affect in this population may also be related to serotonergic disturbance. The 5-HT system has also been found to play an important role in the binge-purge process, albeit in a different way than in restrictors.

Recall that restrictors are proposed to be hyperserotonergic and that acute tryptophan depletion is associated with improved mood. In contrast, acute tryptophan depletion increases depressive mood and mood lability in both ill and recovered BN (Kaye et al., 2000). These results are much more consistent with expectations because tryptophan reduction would subsequently decrease 5-HT levels. Linking these results to binging behavior, consuming high quantities of carbohydrate during a binge may result in an insulin-mediated drop in total plasma LNAA (or large neutral amino acids), reducing competition with tryptophan across the blood-brain barrier, increasing available tryptophan in the brain, accelerating 5-HT release, and eventually, downregulating postsynaptic 5-HT receptors in order to compensate (Kaye & Weltzin, 1991). Alterations in 5-HT activity have been found to persist in

recovered BN (Kaye et al., 1998). Thus, bingeing behavior and food preference may be a direct attempt to regulate mood via neurotransmitter manipulation.

Consistent with this formulation, brain imaging studies using positron emission tomography have found a distinction between AN-R and AN-BP. When compared to recovered AN-R, recovered AN-BP had reduced 5-HT_{2A} receptor activity in the lateral temporal (language), parietal (attention), and occipital (visual) cortical regions (Frank et al., 2002). Additionally, recovered AN-BP had higher 5-HT_{1A} postsynaptic activity in the subgenual cingulate (appetite and sleep) and mesial temporal (memory) regions, as well as increased presynaptic 5-HT_{1A} autoreceptor activity in the dorsal raphe nucleus (largest serotonergic nucleus) area (Kaye et al., 2005). Increased 5-HT_{1A} postsynaptic activity has also been found among patients in the active phase of BN (Tiihonen et al., 2004). These differences suggest enhanced activity in regions of the brain implicated in processing emotional information.

The brain regions associated with bingeing and purging are not specific to AN-BP and BN. Binge-purgers share neurobiological characteristics with other disorders involving dysregulated and/or labile emotion. For instance, the subgenual cingulate region also has been tied to BPD, with this population having increased receptor activity in this region (Siegle, 2007). Furthermore, neuroimaging studies have shown that the subgenual cingulate is overly active in depression (Drevets et al., 1997). The mesial temporal region has been found to be associated with processing emotional autobiographical events (Buchanan, Tranel, & Adolphs, 2006), which may also relate to the preponderance of post-traumatic stress disorder in this population (Swinbourne

& Touyz, 2007). Brain imaging studies further support the neurological link between bingeing-purging and emotion regulation.

Individuals who engage in bingeing and purging are often known to be emotionally labile and generally high in negative affect. Among binge-purgers, the sociocultural model of eating disorders proposes that body dissatisfaction promotes dieting and body dissatisfaction promotes negative affect, which then increases the risk for eating pathology (Crisp, 1984; Stice, 2002). Body dissatisfaction also helps to maintain bulimic symptoms (Stice, 2002), because purging is thought to counter potential weight gain and the negative emotions from bingeing. However, based on biological data, it is likely that trait-related negative affect due to a dysregulated 5-HT system negatively influences one's body self-perception, leading to increased body dissatisfaction.

Binge-Purge Model Summary. Data from a variety of sources (e.g., neuroimaging, self-report, clinical observations) converge to suggest important connections between mood and binge-purge behavior. For instance, it appears that tonic levels of negative affect, rather than acute increases in negative affect, trigger binge eating. However, post-binge mood reports suggest that bingeing is an ineffective way to change mood (Wegner et al., 2002). It is posited within the model that purging has a separate emotion regulatory value. Purging may serve as a way to regulate the negative emotions in general, and to regulate the immediate negative effects of a binge, along with increasing positive affect. The model posits that these behaviors and their emotion regulatory function reinforce one another in a positive feedback

loop. The outcome of the binge-purge cycle is emotionally rewarding; thus, this could account for the chronicity and treatment refractory nature of disorders that rely on bingeing-purging.

Summary

There is a clear link between disordered eating behavior and emotions. Additionally, there is an obvious distinction between individuals with eating disorders who engage in binge/purge behaviors and those who solely restrict their dietary intake. However, exclusive reliance on diagnostic distinctions may obscure these relationships. Most etiological theories posit a sociocultural vulnerability among individuals with eating disorders; however, everyone is subject to images of the thin-ideal, yet not everyone develops AN or BN. Therefore, precipitating individual differences are likely to be present, and it appears many of these pertain to emotion and affect. Once established, eating disorders are difficult to treat. Some individuals resist treatment and fail to recover from eating disorders following multiple intervention attempts. Thus, it can be inferred that the pathological behaviors are serving an emotionally functional purpose in this population. Based on the evidence, it is likely that these affective differences are psychologically and physiologically influenced by a number of factors. Specific disordered eating behaviors, like dietary restriction and bingeing-purging, help to regulate emotions at both psychological and biochemical levels. Thus, the physiological and emotional rewards of the behavior support the chronicity of eating disorders.

Purpose of the Present Study

The present study was a preliminary investigation examining cross-sectional relationships and relied on retrospective recall. Specific attention was given to individuals' temperament (affect intensity), mood (positive affect, negative affect, and depressive symptomatology), and their unique adaptive skills (emotion regulation strategies). This preliminary study was not designed to specify or test directionality; rather, the focus is to emphasize the complicated and multilevel relationships between eating disorder symptoms and different affective constructs. In sum, this model posits an emotion regulation theory of eating disorders.

Hypothesis 1: Behavioral versus Diagnostic Profiles. In line with the model that eating disorders are better distinguished by specific eating disordered behaviors rather than categorical diagnoses, it was hypothesized that emotion dysregulation would be better predicted by behavioral profiles (restrictors, binge-purgers) than by *DSM-IV* diagnostic groups (AN, BN). See Figure 1 for behavioral versus diagnostic model.

See Figure 1, page 87

Hypothesis 2: Affective Constructs in Restrictors and Binge-Purgers. Within the proposed theory, individuals who solely restrict their dietary intake were posited to differ from those who binge-purge on dispositional (or tonic) measures of emotion dysregulation and also in behavior specific (phasic) relationships to affect. Restrictors

were posited to be low in affect intensity, or emotionally constricted. In contrast, binge-purgers were posited to be high in affect intensity, or emotionally labile. Individuals who binge and purge were also posited to experience more depressive symptomatology, more negative affect, and more positive affect than their dietary restrictive counterparts. Figure 2 depicts predicted affective difference between restrictors and binge-purgers.

See Figure 2, page 88

In addition to dispositional measures of emotional dysregulation, it was expected that frequency of disordered eating behaviors and cognitions would have disorder specific relationships to negative and positive affect and would also be related to facets of emotion regulation.

Hypothesis 3: Restricting Behavior and Affective Outcomes. Consistent with expectations that restricting has a functional value in terms of managing emotions, food restriction among restrictors was hypothesized to be a method for increasing positive affect and decreasing negative affect. In addition to restricting behaviors, anorectic cognitions were predicted to be positively correlated with negative affect and inversely related to positive affect. Furthermore, the relationship between emotion regulation skills and outcomes were expected to vary based on affect intensity. Figure 3 depicts predicted correlations between restricting and emotionality.

See Figure 3, page 89

Hypothesis 4: Binge-Purge Behavior and Affective Outcomes. Consistent with the hypothesis that bingeing and purging has a functional value, it was expected that bingeing would be seen as a method for reducing negative affect. Thus, bingeing behaviors would be negatively correlated with negative affect. It was expected that purging behaviors would be positively correlated with both negative affect and positive affect. Finally, the relationship between emotion regulation skills and outcomes were expected to vary based on affect intensity. Figure 4 depicts predicted correlations between disordered eating behaviors and emotionality among binge-purgers.

See Figure 4, page 90

CHAPTER II

METHOD

Participants

Participants were 63 female inpatients at the Research Medical Center's VITA Eating Disorders Program, mean age 31.9 years ($SD = 11.77$). The majority were Caucasian (95.2%). Participants' average body mass index (BMI) was 20.2 ($SD = 7.62$). BMI was calculated by the following formula: weight in kilograms / height in meters². Twenty-two participants met criteria for AN-R (34.9% dietary restrictors). Twelve participants met criteria for AN-BP and 29 participants met criteria for BN (65.1% binge-purgers). A total of 72 patients were given the opportunity to participate; 87.5% completed all or most of the questionnaire packet and 12.5% chose not to participate. Of the non-participants, two (22.2%) were diagnosed with AN-R, two (22.2%) with AN-BP, and five (55.6%) with BN. See Table 1 for complete demographic characteristics.

See Table 1, page 81

Measures

Diagnostic Classification. The Eating Disorder Diagnostic Scale (EDDS; Appendix A; Stice, Telch, & Rizvi, 2000) is a 22-item self-report scale that can be used to diagnose AN and BN. Items from the EDDS are designed to assess *DSM-IV* diagnostic criteria for eating disorders. The first four items assess the individuals'

thoughts surrounding their weight and shape using a seven-point Likert Scale [e.g., “Has your weight influenced how you think about (judge) yourself as a person?”]. The next 14 questions assess the presence of bingeing and purging behavior [e.g., “During the times when you ate an unusually large amount of food, did you experience a loss of control (feel you couldn’t stop eating or control what or how much you were eating)? YES/NO] and the frequency of eating disorder behavior questions (e.g., “How many times per week on average over the past 3 months have you made yourself vomit to prevent weight gain or counteract the effects of eating?”). The last four items assess height/weight and menstrual status. The EDDS was used in two ways, as a measure of symptom severity and also as a diagnostic tool. Psychometric information for both applications is presented below.

This scale has been determined to be both reliable and valid as a continuous measure of symptom severity. The internal consistency of the EDDS symptom composite was found to be .91 across ED diagnostic groups (Stice, Telch, & Rizvi, 2000). The 1-week test-retest kappa coefficient was .95 for AN diagnoses and .71 for BN diagnoses (Stice, Telch, & Rizvi, 2000). Thus, the EDDS has been shown to be an internally consistent and stable measure of the degree of restricting, bingeing, and purging behavior. The internal consistency of the EDDS symptom composite in the total present sample was $\alpha = .68$. When groups were split according to *DSM-IV* diagnosis, internal consistency among those classified as AN was $\alpha = .71$ and for BN was $\alpha = .56$.

In addition to assessing symptom severity, the EDDS was used to assign participants to diagnostic groups. Importantly, there is a high degree of concordance between the EDDS and the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993), the “gold standard” in clinical diagnosis of eating disorders. The EDDS correctly identified 98% of women with AN and 91% with BN (Stice, Telch, & Rizvi, 2000). Furthermore, the correlation between the EDDS symptom composite and the EDE symptom composite was .82 (Stice, Fischer, & Martinez, 2004).

The diagnostic classification scheme used in this study follows published norms for the EDDS (Stice, Telch, & Rizvi, 2000). A participant was assigned to the restrictor group if she reported: (a) height and weight data on EDDS Items 19 and 20 that resulted in a BMI of less than 17.5, (b) a fear of weight gain or becoming fat as indexed by a score of 4 or greater on EDDS Item 2, (c) undue influence of body weight or shape on self-evaluation as indexed by a score of 4 or greater on either EDDS Item 3 or 4, and (d) amenorrhea in postmenarcheal females as indexed by a 3 on EDDS Item 21. Following the EDDS scoring algorithm, if an individual met the first and fourth criteria above, it was not necessary for the individual to endorse the second and third criteria. Further, because oral contraceptives can result in a regular menstrual cycle, participants who were taking oral contraceptives that met the low weight criteria were coded as amenorrheic. Restrictors were excluded from this group if they responded *yes* to either Item 5 or 6. These criteria were designed to identify dietary restrictors who do not engage in binge-purge behaviors.

Subthreshold AN on the EDDS is assigned if an individual meets all aforementioned criteria except for the low BMI criterion (i.e., their BMI falls between 17.5 and 19). There were five individuals with AN-R that fell within this category in the current sample. For the purpose of this study and to maintain power, these individuals were classified as restrictors. It is important to note that according to the proposed criteria for the newest version of APA's diagnostic manual (*DSM-5*; APA, 2010), these "subthreshold" individuals would meet full criteria for AN in the future. The new criteria specify, "...a weight that is less than minimally normal," with a new 'Severity' index where an individuals' BMI is taken into account.

Membership in the binge-purge group was assigned to women who reported: (a) regular eating binges marked by a perceived loss of control and the consumption of a large amount of food as indexed by a response of *yes* to EDDS Item 5, a *yes* to EDDS Item 6, and a response of greater than or equal to 2 on EDDS Item 8; (b) regular use of compensatory behaviors as indexed by a response of 8 or greater on the sum of EDDS Items 15, 16, 17, and 18; and (c) undue influence of body weight or shape on self-evaluation as indexed by a score of 4 or greater on either EDDS Item 3 or 4.

Subthreshold BN on the EDDS is assigned if an individual meets the aforementioned criteria but binges and/or purges once per week as opposed to the more stringent twice per week as defined by the *DSM-IV*. There were three individuals that fell within this category in the current sample. Additionally, there were five individuals with AN-BP that fell into the subthreshold AN category on the

EDDS. For the purpose of this study, all of these individuals were classified as binge-purgers. Again, it is important to note that according to the proposed criteria for the *DSM-5* (APA, 2010) these “subthreshold” individuals would meet full criteria for AN-BP and BN in the future. The new criteria for BN will include the ‘Severity’ index where the frequency of episodes is taken into account.

Affect Intensity. The Affect Intensity Measure (AIM; Appendix B; Larsen & Diener, 1987) is a 40-item self-report questionnaire that assesses how strongly an individual experiences their emotions. Using a six-point Likert Scale, items assess both positive emotional reactions (e.g., “When I accomplish something difficult I feel delighted or elated”) and negative emotional reactions (e.g., “The sight of someone who is hurt badly affects me strongly”). Higher scores reflect higher affect intensity. Internal consistency for the AIM ranges between .90 and .94 (Larsen, 1984). Test-retest reliabilities at 1-, 2-, 3-month, and 2-year intervals are .80, .81, .81, and .75, respectively (Larsen, 1984). Observed internal consistency for the present sample was $\alpha = .87$.

Affect. The Positive and Negative Affect Schedule (PANAS; Appendix C; Watson, Clark, & Tellegen, 1988) is a self-report measure used to assess two (relatively) uncorrelated dimensions of affect: positive affect (PA) and negative affect (NA). PA is defined as the extent to which one feels excited, active, and alert. NA measures the extent in which one feels subjective distress, displeasure, and anger. Items are rated on a five-point Likert Scale. PA and NA subscales are formed by averaging the ratings of PA and NA items. Watson and colleagues’ (1988) published

psychometric data show that the PANAS is internally consistent, stable, and that the subscales correlate in the expected directions with related constructs. Internal consistency has been observed to range from .86 to .90 for PA and from .84 to .87 for NA. Test-retest reliabilities with an 8-week retest interval are .58 and .48 for PA and NA, respectively. Construct validity for the PANAS has been shown by correlating it with the Hopkins Symptom Checklist (HSCL), the Beck Depression Inventory (BDI), and the State-Trait Anxiety Inventory State Anxiety Scale (A-State). In this study, participants were asked to recall to what extent they experienced PA and NA: over the course of one week (PANAS-W), when they restricted food intake (PANAS-R), following an episode of bingeing (PANAS-B), and after purging (PANAS-P). Observed internal consistency for the present sample on the PANAS-W was $\alpha = .90$ for PA and $\alpha = .88$ for NA.

Emotion Regulation. The Emotion Amplification and Reduction Scale (TEARS; Appendix D; Hamilton et al., 2009) is a measure used to assess people's ability to regulate their emotions. It is designed to assess perceived ability to change the trajectory of an emotional response. Amplification items assess an individual's ability to prolong or intensify an existing emotion (e.g., "If I wanted to, I could turn UP the intensity level of whatever emotion I may be feeling"). Reduction items assess an individual's ability to select an emotional response or alter an existing emotion by softening, shortening, or stopping an existing emotion (e.g., "I can stop an emotion before it overwhelms me"). Amplification items were found to correlate with higher PA, higher NA, and greater fatigue. Reduction items were found to correlate with less

depression, less fatigue, and less NA. TEARS has been found to have good internal consistency (emotion amplification $\alpha = .87$ and emotion reduction $\alpha = .89$) (Hamilton et al., 2009). Observed internal consistency for the present sample was $\alpha = .85$ for emotion amplification and $\alpha = .84$ for emotion reduction.

Symptoms of Depression. The Beck Depression Inventory (BDI; Beck et al., 1961) is a self-administered, 21-item self-report measure used to assess depressive symptomatology. Participants indicate to what degree certain symptoms of depression apply to them (e.g., “I do not feel sad; I feel sad much of the time; I am sad all the time; I am so sad or unhappy that I can’t stand it”). Internal consistency ranges from .73 to .92 with a mean of .86 (Beck, Steer, & Garbin, 1988). The BDI has a high internal consistency (psychiatric population $\alpha = .86$ and non-psychiatric population $\alpha = .81$) (Beck et al., 1988). Test-retest reliabilities range from .48 to .86, and are dependent upon the interval between re-testing and the nature of the population (Groth-Marnat, 1990). Observed internal consistency for the present sample was $\alpha = .93$.

Procedure

Inpatients at the Research Medical Center’s VITA Eating Disorders Program were offered the opportunity to participate in this study. Upon intake at the hospital, the intake nurse gave potential participants a manila envelope containing a statement of informed consent and the questionnaire packet. The statement of informed consent (Appendix E) explained the purpose of the study and asked for participation. Participants indicated consent by returning an unsigned statement of informed

consent and a completed questionnaire packet. The participants were instructed to keep one copy of the informed consent statement. The statement informed participants that there was no penalty for non-participation and that the staff at Research Medical Center would not know whether or not they chose to participate in the study. The questionnaire packet consisted of a recruitment letter with instructions (Appendix F), a demographic questionnaire (Appendix G), and the aforementioned measures.

The assessment battery took approximately 30 minutes to complete. Once a consenting participant completed the assessment battery, she sealed one statement of informed consent and the completed questionnaires inside the envelope and returned it to the intake nurse. She kept the other informed consent statement for herself. At that time, the intake nurse wrote the participant's BMI and *DSM-IV* diagnosis/es on the outside of the sealed envelope. Participants who did not wish to complete the questionnaires were instructed to seal the blank questionnaire and the unsigned consent form in the sealed manila envelope and return it to the intake nurse. This procedure ensured that the staff at Research Medical Center remained unaware of who chose to participate and also allowed tracking of consenting and non-consenting participants. Each participant was assigned a unique identification number and was identified by that number in all data files. Participants were not asked to divulge their names or other identifying information. Participants did not sign statements of informed consent. Thus, no specific identifying information about the participant was known.

Data Analysis

Data were analyzed utilizing the following statistical techniques: analyses of covariance (ANCOVA), ordinary least squares (OLS) regression, tests of correlation (Pearson's product-moment coefficient [r]), and independent samples t-tests.

Overarchingly, it was hypothesized that women classified as restrictors versus binge-purgers would show different patterns of emotional processing. Missing data (3.02%) were imputed in LISREL 8.7 using estimated means and five iterations. All other analyses were conducted utilizing SPSS 18. A correlation matrix (see Table 2) was used to identify possible covariates of emotionality. Thus, any relevant analyses covaried for age.

See Table 2, page 82

CHAPTER III

RESULTS

Power Analysis

A post hoc power analysis was completed using the obtained sample size ($N = 63$) and desired effect size (0.40) to determine the power of the chosen analyses. This presumes that the effect size in the sample is equal to the effect size in the general disordered eating population. The power analysis revealed that, in order to have sufficient power to find large effect sizes with an alpha of .05, a total of 42 participants – 21 participants in each of the two groups – were necessary. Based on these parameters, this sample had adequate power. Full results of the power analysis are presented in Appendix H.

Hypothesis 1: Behavioral versus Diagnostic Profiles

In line with the model that eating disorders are better distinguished by specific eating disordered behaviors rather than categorical diagnoses, it was hypothesized that emotion dysregulation is better predicted by behavioral profiles (restrictors, binge-purgers) than by *DSM-IV* diagnostic groups (AN, BN).

Profile Comparison. Use of the behavioral and *DSM-IV* diagnostic profiles produced significantly different groups, $\chi^2(1, N = 63) = 28.84, p < .01$. The *DSM-IV* diagnostic scheme classified 34 participants as AN and 29 participants as BN. In contrast, the behavioral scheme classified 22 participants as restrictors and 41 as binge-purgers. The discrepancies between the models were exclusively in diagnostic classification of AN. Of the 34 patients classified as AN, the behavioral model

classified 22 as restrictors and 12 as binge-purgers. In contrast, of the 29 participants classified as BN, all 29 were classified as binge-purgers. Given the degree of overlap between the two models (and thus colinearity) for the BN diagnosis, the relationships to affective outcomes were considered separately for each diagnostic scheme. A series of one-factor ANCOVAs were used to determine whether classification schemes predicted affective outcomes (see Table 3). All analyses followed the same format: classification scheme (either behavioral: restrictors versus binge-purgers or *DSM-IV*: AN versus BN) was entered as a fixed effect, and age was entered as a covariate where appropriate.

Affect Intensity. The behavioral, but not the diagnostic classification scheme, was associated with marginal differences in affect intensity (AIM). Consistent with predictions, binge-purgers ($M = 148.54$, $SD = 23.72$) had marginally higher levels of affect intensity than did restrictors ($M = 136.95$, $SD = 21.08$), $F(1, 61) = 3.68$, $p = .060$, partial $\eta^2 = .057$, observed power = .47. In contrast, there was not a significant affect intensity difference between AN ($M = 141.09$, $SD = 25.84$) and BN ($M = 148.48$, $SD = 19.70$), $F(1, 61) = 1.59$, $p = .213$, partial $\eta^2 = .025$, observed power = .24. Age was not correlated with affect intensity and thus was not used as a covariate in either analysis.

Depressive Symptomatology. Neither the behavioral nor the *DSM-IV* diagnostic schemes were related to differences in level of depressive symptoms (BDI).

Weekly Negative Affect. Neither the behavioral nor the *DSM-IV* diagnostic schemes were related to differences in level of NA (PANAS-W).

Weekly Positive Affect. Neither the behavioral nor the *DSM-IV* diagnostic schemes were related to differences in level of PA (PANAS-W).

See Table 3, page 83

Hypothesis 2: Affective Constructs in Restrictors and Binge-Purgers

Within the proposed theory, individuals who solely restrict their dietary intake were posited to differ from those who binge-purge in dispositional indices of emotion dysregulation. Restrictors were posited to be low in affect intensity, or emotionally constricted. In contrast, binge-purgers were posited to be high in affect intensity, or emotionally labile. Individuals who binge and purge were also posited to experience more depressive symptomatology, more negative affect, and more positive affect than their restricting counterparts. Finally, individuals who binge and purge were hypothesized to be higher in emotion amplification and lower in emotion reduction than the dietary restrictors.

Tonic differences. As reported above, binge-purgers had marginally higher affect intensity than restrictors ($p = .06$), but there were no other significant differences between restrictors and binge-purgers in terms of depressive symptomatology, emotion amplification, or emotion reduction.

Phasic Differences. As shown in Table 4, restrictors reported significantly more PA post-purge than binge-purgers (mean difference = 9.765). Although dietary restrictors do not binge, they often endorse purging via vomiting (e.g., after eating one cookie) and over-exercising in an attempt to lose more weight. Therefore, a number of the restricting participants ($n = 17$) completed the PANAS-P measure based on these experiences, allowing for comparison of this behavior. There were no other group differences in affect following disordered eating behaviors.

See Table 4, page 84

Hypothesis 3: Restricting Behavior and Affective Outcomes

Food restriction in restrictors was hypothesized to be a method for increasing positive affect and decreasing negative affect. Specifically, dietary restriction was proposed to be associated with constricted range and intensity of emotion (AIM), diminished ability to regulate emotion (TEARS-Reduction), more depressive symptomatology (BDI), and more NA and PA (PANAS). In addition to restricting behaviors, anorectic cognitions were predicted to be positively correlated with indices of emotion dysregulation. Furthermore, individuals' dispositional levels of emotionality were proposed to moderate these relationships.

Correlational Analyses for Restrictors. Tests of correlation (Pearson's product-moment coefficient [r]) were used to test the relationship between restricting eating behavior and indices of affect and emotion among those who solely restrict

(see Table 5). Consistent with the model, there was a positive relationship between restricting frequency and symptoms of depression, $r(21) = .51, p < .05$. However, there was no relationship between restricting frequency and other affective outcomes including: affect intensity, weekly PA and NA, PA and NA following restriction, or dimensions of emotion regulation.

There were significant relationships between anorectic cognitions and affective outcomes. As shown in Table 5, feeling fat was related to higher levels of weekly NA. Fear of fat was related to higher levels of depressive symptomatology and weekly NA. Additionally, using weight and shape for evaluative purposes negatively covaried with the ability to regulate emotions by way of emotion reduction.

See Table 5, page 85

Emotion Regulation and Response to Restricting. OLS regression was used to examine whether there was a relationship between emotion regulation and affect following dietary restriction and whether that relationship was moderated by affect intensity. Following procedures outlined by Aiken and West (1991), variables were centered and interactions were plotted using an interactive web program (Preacher, Curran, & Bauer, 2006).

Emotion Reduction and PA Post-Restriction. Post-restriction PA was related to the interaction between affect intensity and emotion reduction. There was no first

order relationship between emotion reduction and PA ($\beta = -.378$, $SE = .439$, $p = .401$), nor was there a relationship between affect intensity and PA post-restriction ($\beta = .148$, $SE = .110$, $p = .196$). However, there was a significant interaction ($\beta = .048$, $SE = .018$, $p = .018$). Increasing levels of emotion reduction were related to low levels of PA post-restriction for those with low levels of affect intensity ($\beta = -1.390$, $t = -2.824$, $p = 0.011$). However, there was no relationship between emotion reduction and positive affect post-restriction for those with either high levels of affect intensity ($\beta = 0.634$, $t = 1.110$, $p = 0.282$) or those with average levels of affect intensity ($\beta = -.378$, $t = -0.972$, $p = 0.344$). These data suggest that restriction is related to lower levels of positive affect post-restriction only for those restrictors who have the most constricted affective systems, those with the lowest affect intensity and highest levels of emotion reduction (see Figure 5).

Emotion Reduction and NA Post-Restriction. NA was related to affect intensity in the expected direction. There was a first order relationship between affect intensity and NA post-restriction ($\beta = .250$, $SE = .086$, $p = .009$). However, there was no first order relationship between emotion reduction and NA ($\beta = .087$, $SE = .342$, $p = .802$), nor was there an interaction ($\beta = -.019$, $SE = .116$, $p = .260$). Consistent with theories of emotion regulation, restrictors who were high in affect intensity reported significantly more NA post-restriction compared to restrictors who were low in affect intensity.

Emotion Amplification and PA Post-Restriction. No main effects were found regarding affect intensity and emotion amplification for PA after restricting, nor was there an interaction effect.

Emotion Amplification and NA Post-Restriction. As noted above, NA-post restriction covaried with affect intensity ($\beta = .244$, $SE = .019$, $p = .021$). However, there was no first order relationship between emotion amplification and NA ($\beta = .009$, $SE = .347$, $p = .980$), nor was there an interaction ($\beta = .010$, $SE = .015$, $p = .497$). Similar to the above findings, restrictors who were high in affect intensity reported significantly more NA post-restriction. This relationship holds true regardless of one's specific emotion regulation strategies (amplification or reduction).

See Figure 5, page 91

Hypothesis 4: Binge-Purge Behavior and Affective Outcomes

It was hypothesized that bingeing would be seen as a method for reducing negative affect. The model led to the predictions that frequency of bingeing-purging would be associated with a variable and intense range of emotion (AIM), diminished ability to regulate emotions (TEARS), and more depressive symptomatology (BDI). Restricting behaviors in this population were expected to be associated with more NA and PA (PANAS-R). It was also predicted that bingeing behavior would be correlated with higher levels of NA and lower levels of PA (PANAS-B), and be related to emotion amplification (TEARS-Amplification). Finally, it was predicted that purging

behavior would be correlated with lower levels of NA and higher levels of PA (PANAS-P), and be related to emotion amplification (TEARS-Amplification).

Correlational Analyses for Binge-Purgers. Tests of correlation (Pearson's product-moment coefficient [r]) were used to test the relationship between eating disordered behavior and indices of affect and emotion among those who binge and purge (see Table 6). No relationship was found between frequency of bingeing-purging and depressive symptomatology, affect intensity, or PA. Interestingly and inconsistent with the model, more frequent bingeing was related to lower NA post-restriction, $r(40) = -.34, p < .05$. Alternatively and consistent with the model, purging behaviors (laxative abuse, over-exercise) were positively related to emotion amplification abilities. Over-exercising was related to higher levels of weekly NA and also higher levels of PA following a purge. Additionally, significant relationships between bulimic cognitions and affective outcomes were found. Specifically, individuals who use shape for evaluative purposes reported more weekly NA and individuals who use weight for evaluative purposes reported greater emotion amplification abilities.

See Table 6, page 86

Emotion Regulation and Response to Restricting, Binging, and Purging. OLS regression was used to examine whether affect intensity moderated the relationship between emotion regulation and PA/NA following disordered eating behaviors in binge-purgers. Following procedures outlined by Aiken and West (1991), variables

were centered and interactions were plotted using an interactive web program (Preacher, Curran, & Bauer, 2006).

Emotion Amplification and PA Post-Restriction. Emotion amplification was the sole predictor of post-restriction PA. There was a first order relationship between emotion amplification and PA post-restriction ($\beta = .709$, $SE = .305$, $p = .027$). However, there was no first order relationship between affect intensity and PA ($\beta = -.054$, $SE = .069$, $p = .439$), nor was there an interaction ($\beta = .021$, $SE = .019$, $p = .268$). Thus, binge-purgers who were high in emotion amplification abilities reported significantly more PA post-restriction than binge-purgers low in emotion amplification abilities.

Emotion Amplification and NA Post-Restriction. No main effects were found regarding affect intensity and emotion amplification for NA after restricting, nor was there an interaction effect.

Emotion Reduction and PA Post-Restriction. No main effects were found regarding affect intensity and emotion reduction for PA after restricting, nor was there an interaction effect.

Emotion Reduction and NA Post-Restriction. No main effects were found regarding affect intensity and emotion reduction for NA after restricting, nor was there an interaction effect.

Emotion Amplification and PA Post-Binging. Post-binge PA was dependent upon the interaction between emotion amplification and affect intensity. Although there was no first order relationship between affect intensity and PA post-binge ($\beta = -$

.011, $SE = .047$, $p = .812$), there was a first order relationship between emotion amplification and PA ($\beta = .792$, $SE = .208$, $p = .001$). This relationship, however, was moderated by one's affect intensity ($\beta = .024$, $SE = .012$, $p = .041$). For those high ($\beta = 1.367$, $t = 5.803$, $p < .01$) or average in affect intensity ($\beta = 0.792$, $t = 3.694$, $p < .01$), higher levels of emotion amplification were related to higher levels of post-binge PA. However, increasing levels of emotion amplification were not related to PA post-binge for those low in affect intensity ($\beta = 0.217$, $t = 0.558$, $p = 0.580$). These data suggest that for binge-purgers, increasing levels of emotion amplification along with average to high levels of affect intensity were associated with more PA after bingeing (see Figure 6).

Emotion Amplification and NA Post-Binging. No main effects were found regarding affect intensity and emotion amplification for NA after bingeing, nor was there an interaction effect.

Emotion Reduction and PA Post-Binging. Ability to reduce emotions was related to higher PA post-binge ($\beta = .616$, $SE = .193$, $p = .003$). However, there was no first order relationship between affect intensity and PA ($\beta = .062$, $SE = .051$, $p = .232$), nor was there an interaction ($\beta = .004$, $SE = .007$, $p = .604$). Thus, for binge-purgers, emotion reduction covaried with PA.

Emotion Reduction and NA Post-Binging. Post-binge NA was dependent upon the interaction between emotion reduction and affect intensity. Although there was no first order relationship between affect intensity and NA post-binge ($\beta = .053$, $SE = .052$, $p = .315$), there was a marginal first order relationship between emotion

reduction and NA ($\beta = -.399$, $SE = .197$, $p = .050$). This relationship, however, was moderated by one's affect intensity ($\beta = .016$, $SE = .007$, $p = .026$). For those low in affect intensity ($\beta = -0.782$, $t = -3.223$, $p = .003$) or average in affect intensity ($\beta = -0.399$, $t = -2.132$, $p = 0.040$), higher emotion reduction levels were associated with lower levels of NA post-binge. In contrast, increasing levels of emotion reduction were not related to NA post-binge for those high in affect intensity ($\beta = -0.016$, $t = -0.059$, $p = 0.953$). These data suggest that for binge-purgers, increasing levels of emotion reduction along with low to average to levels of affect intensity were associated with less NA after bingeing (see Figure 7).

Emotion Amplification and PA Post-Purging. There was a first order relationship between emotion amplification and PA post-purge ($\beta = 1.196$, $SE = .277$, $p < .01$). However, there was no first order relationship between affect intensity and PA ($\beta = -.028$, $SE = .063$, $p = .655$), nor was there an interaction ($\beta = .018$, $SE = .016$, $p = .267$). Thus, binge-purgers who were high in emotion amplification abilities reported significantly more PA post-purge than binge-purgers who were low in emotion amplification abilities.

Emotion Amplification and NA Post-Purging. No main effects were found regarding affect intensity and emotion amplification for NA after purging, nor was there an interaction effect.

Emotion Reduction and PA Post-Purging. No main effects were found regarding affect intensity and emotion reduction for PA after purging, nor was there an interaction effect.

Emotion Reduction and NA Post-Purging. No main effects were found regarding affect intensity and emotion reduction for NA after purging, nor was there an interaction effect.

See Figures 6 and 7, pages 92-93

CHAPTER IV

DISCUSSION

Summary of Findings

Results of this study indicated that emotionality does indeed play a role in eating disorders. Although it is customary to use the *DSM-IV* diagnostic scheme, the results of this study suggest that it may be more useful to divide eating disorder groups based on behavioral differences. There were differences between restrictors and binge-purgers in: relationships of disordered eating behaviors and affective outcomes at dispositional levels, the way in which eating disorder behaviors and cognitions related to affective outcomes, and affective processing. In summary, the relationship between eating disorders and emotion is complex and multifaceted and appears to differ in those with restricting versus binge-purge disorders.

Differences between Restrictors and Binge-Purgers

Consistent with study hypotheses, binge-purgers reported marginally higher levels of affect intensity than did restrictors. Restrictors are known to be uncomfortable with both the feeling and expression of strong affect (Vitousek & Ewald, 1993), whereas binge-purgers are more likely to be volatile and emotionally labile (Westen & Harnden-Fischer, 2001; Vitousek & Manke, 1994). So the dispositional difference found in the present study is not surprising. Although there was no between group difference in post-restriction affect, groups did differ in post-purge affect. Restrictors endorsed significantly more positive affect post-purge (subjective purging) than binge-purgers reported after purging. Although between

group differences were not as extensive as expected, the results regarding affect intensity are in line with previous research.

Within Group Differences

The model tested here suggested that in addition to dispositional affective differences, disordered eating behaviors and cognitions would have functional relationships to affect and would also be related to facets of emotion regulation. In restrictors, food restriction was hypothesized to be a method for increasing positive affect and decreasing negative affect. Among those who binge and purge, it was hypothesized that bingeing would be seen as a method for reducing negative affect and that purging behaviors would increase both negative affect and positive affect.

Restrictors. The model tested suggested that food restriction would increase positive affect and decrease negative affect and that the relationship between emotion regulation skills and outcomes would vary based on affect intensity (Figure 3). Among restrictors, there was a positive relationship between restricting frequency and symptoms of depression. Although directionality cannot be determined from these data, the model proposed in this study would suggest that symptoms of depression trigger restricting behaviors. In addition, there were significant relationships between anorectic cognitions and affective outcomes. Specifically, feeling fat was correlated with higher levels of tonic (weekly) negative affect and fear of becoming fat was related to both depressive symptomatology and negative affect. Furthermore, individuals who used weight and shape for evaluative purposes reported more limited abilities downregulating their emotions. Thus, more severe negative anorectic

cognitions were associated with more depressed mood and constrained ability to downregulate dysphoric affect and dysphoric mood, in turn triggering restricting behavior. This is congruent with the sociocultural model of eating disorders (Garner, Olmstead, & Polivy, 1983; Stice, 2002). Consistent with what was found here, when individuals internalize the thin ideal and place too much value on the importance of appearance, body dissatisfaction increases, which in turn promotes dieting, negative affect, and eating pathology.

Importantly, there appeared to be individual differences in the relationship between emotion regulation and affective outcomes. The results are largely consistent with emotion regulation theories suggesting that there are (at least) two dimensions of emotional processing; dispositional intensity levels and the ability to control resulting feelings (Derryberry & Rothbart, 1988). Consistent with this, those low in affect intensity and high in emotion reduction abilities reported less post-restriction positive affect (Figure 5). These restrictors could be thought of as being at the extreme end of an already constricted affective range.

In contrast, restrictors with a greater affective range (higher in affect intensity) do not appear to benefit from this increased range. Restrictors who were high in affect intensity reported significantly more post-restriction negative affect compared to restrictors who were low in affect intensity. This relationship held true regardless of one's specific emotion regulation strategies. This suggests that restrictors do not have the capacity to downregulate or reduce stronger negative emotions, a clear reflection of their difficulties recognizing and regulating emotions (Harrison et al., 2009). In the

subpopulation of restrictors highest in dispositional affect intensity, perhaps learning to better regulate emotions could have a significant effect on tonic emotionality. This is especially relevant based on the finding that dietary restriction is not effective in managing one's mood.

Binge-Purgers. The model tested here proposed that bingeing would be seen as a method for repairing negative affect after periods of dietary restriction, and that purging behaviors would be reparative, increasing both negative affect and positive affect (Figure 4). Furthermore, the relationship between emotion regulation skills and outcomes was expected to vary based on affect intensity. In contrast from the model proposed here, frequency of bingeing was related to lower post-restriction negative affect. The model would suggest that increasing levels of negative affect due to ongoing restriction would leave one vulnerable to bingeing; however, the data demonstrate the opposite relationship. This could in actuality be true, or conversely, self-reports could be tainted by retrospective bias. Individuals who frequently binge could idealize times in which they were able to restrict dietary intake and forget the toll that dietary restriction takes on them both physiologically and psychologically. These results emphasize the necessity of measuring affect in closer temporal proximity to the behavior in question.

Binge-purgers were expected to have difficulty downregulating their emotions and more negative affect associated with their disordered eating behavior. This was in part supported by the finding that the frequency of purging behaviors was positively

related to emotion amplification abilities and more negative affect over the course of a week. Thus, purging appears to be associated with a variety of affective outcomes.

Significant relationships between bulimic cognitions and affective outcomes were also found. Similar to restrictors, binge-purgers who used weight for evaluative purposes reported more negative affect over the course of a week. This is consistent with the notion that body dissatisfaction promotes negative affect, which then increases the risk for eating pathology (Crisp, 1984). In contrast to restrictors, binge-purgers who used shape for evaluative purposes reported greater abilities upregulating emotions. This may mean that binge-purgers can use shape cues to increase feelings of positive affect, perhaps by utilizing downward social comparison (Wills, 1981). Thus, the way in which one feels about one's body has an important influence on affective outcomes.

As with restrictors, it appears that there are individual differences in the relationship between emotion regulation and affective responses to disordered eating behaviors that are masked by affect intensity. Binge-purgers who were high in both affect intensity and emotion amplification abilities (the most emotionally extreme subgroup) experienced the most positive affect after bingeing (see Figure 6). The opposing subgroup – binge-purgers low in affect intensity and high in emotion reduction abilities (the most emotionally constricted subgroup) – experienced the least negative affect after bingeing (see Figure 7). Consistent with the model proposed here, binge-purgers with high affect intensity and abilities upregulating emotions appear to be able to capitalize on positive emotions. Moreover, binge-purgers with

greater abilities downregulating emotions experienced greater positive affect after bingeing as well. This is consistent with the affect regulation model which posits that individuals binge eat to provide relief and distraction from unpleasant emotions (McCarthy, 1990). Accordingly, emotion regulation abilities (both up- and downregulation) reliably predicted positive affect after bingeing. Perhaps being able to successfully regulate emotions is adjunctively mood protective following a binge.

Similar to affective consequences of bingeing, responses to restricting and purging were also related to emotion regulation skills among binge-purgers. Consistent with the model, binge-purgers who were better equipped to regulate emotions seem to be able to exploit positive emotions resulting from successfully restricting food intake or “getting rid” of excess calories via purging. This is in line with research findings that purport purging to be emotionally cathartic (Hawkins & Clement, 1984). Effective emotion regulation strategies primarily served to increase positive affect after restricting, bingeing, and purging as well as decrease negative affect after bingeing. Thus, one’s regulatory abilities reliably produced positive emotional outcomes due to disordered eating. This highlights the affectively reinforcing component of disordered eating in this population and emphasizes the importance of elucidating these complex relationships.

Future Directions

The results of this study suggest that it would be important to continue investigation into the role that emotions play in the development, maintenance, and treatment of eating disorders. Specifically, further examination of the neurobiological

underpinnings of eating and emotion may provide greater insight into these complicated relationships. Clearly, there is a strong relationship between eating and emotions. Emotional vulnerability and maintenance of eating disorder pathology may also be bolstered by neurobiology. Dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis is well documented in the eating disordered population (Stokes & Sikes, 1991). The HPA axis is responsible for both feeding behaviors and emotional functioning. Tracts of neurons within the HPA axis control a number of neurotransmitters and neuromodulators including those with known relationships to eating disorders. Neuroscience may help us better understand this complicated and dynamic relationship and illuminate the role of emotions in disordered eating behavior. Utilizing studies on the role of 5-HT as well as neuroimaging data can help bridge the gap between self-report, observational, and biological data. More specifically, neuroimaging results and blood serum 5-HT levels provide measurements of state-dependent activity, linking neural events, emotional reactions, and behavioral responses.

Results from the present study suggest differences between individuals that binge-purge and those that solely restrict their dietary intake. Furthermore, these behaviors were found to play a role in emotion regulation. Gross's (1998a, 1998b, and 2001) process model of emotion regulation may help elucidate these processes. He posits that the emotional timeline is broken down into antecedent-focused and response-focused emotion regulation strategies (Gross, 1998b). Response modulation refers to attempts by an individual to influence behavioral and physiological

responses once an emotion is already in motion (e.g., emotional suppression) (Gross, 1998b). In contrast, antecedent-focused approaches refer to prophylactic attempts to control emotion before it is fully activated (e.g., cognitive reappraisal). Antecedent-focused emotion regulation strategies have been found to decrease experiential, behavioral, and physiological responses, whereas response-focused strategies have been found not to change the emotional experience and to actually increase physiological responses (Gross, 1998a). Additionally, in emotionally negative situations, antecedent-focused strategies decrease negative emotion-expressive behavior but do not change positive emotion-expressive behavior (Gross, 1998a). On the other hand, response-focused strategies decrease both negative and positive emotion-expressive behavior (Gross, 1998a). In sum, differential emotion outcomes and trajectories are based on when and how one modulates a specific emotion. Based on the present study's findings, it may be that restrictors are more likely to use antecedent-focused strategies, whereas binge-purgers are more likely to use response-focused strategies.

In a recent fMRI study that examined the neural bases of emotion regulation in a healthy population, interesting differences were found between the emotion regulatory mechanisms of antecedent- and response-focused emotion regulation strategies (Goldin, McRae, Ramel, & Gross, 2008). Cognitive reappraisal, an antecedent-focused strategy, resulted in increased activity in the cognitive control areas of the prefrontal cortex and decreased activation in the amygdala and insula. On the other hand, emotional suppression, a response-focused strategy, resulted in

sustained elevations in the amygdala and insula. Reappraisal and suppression produced differential effects on emotional experience and opposing effects on the neural response in two key areas of the limbic system.

These neurobiological indices of emotion regulation would be interesting to explore in a disordered eating population. 5-HT has known relationships to both feeding and emotional behavior; therefore, it is not surprising that dysregulation in this system is evident among individuals with an eating disorder (see Kaye, 2008 for review). Regarding functional imaging data, there is an overlap between brain regions that are activated in tests of emotion regulation in healthy populations and those activated in studies on eating disordered patients. Areas active during the regulation of emotion, like the prefrontal and cingulate cortices (Ochsner, 2007), would be interesting to explore among those with an eating disorder. More specifically, these areas may have more or less activation in those with restricting versus binge-purge behaviors. Examination of affective processes in vivo could further an understanding of the emotion regulation theory of eating disorders.

Treatment Implications

Results of this study may prove clinically useful. Effective treatments for disordered eating are few and far between (see Treasure, Claudino, & Zucker, 2010 for review). Perhaps treatments aimed specifically at affective components of dysregulated eating would be particularly suited to combat these difficult to treat disorders. Results from the present study suggest that individuals with eating disorders utilize food and maladaptive behaviors to regulate their moods. Teaching

skills aimed at downregulating negative affect and upregulating positive affect without the use of these maladaptive behaviors may be a way to combat disordered eating.

Dialectical Behavior Therapy (DBT) has been adapted for use in binge-purge disorders and specifically focuses on emotion regulation strategies (Safer, Telch, & Agras, 2001). This treatment aims to replace bingeing and purging with emotion regulation skills, and preliminary studies have shown promising results. Moreover, DBT has been shown to eradicate binge eating in up to 89% of women with binge eating disorder by implementing mindfulness, emotion regulation, and distress tolerance skills (Telch, Safer, & Linehan, 2001). Perhaps modalities similar to these could be modified to target alexithymia and constricted emotionality in dietary restrictors as well.

One of the most successful and robust treatments of eating disorders to date is Cognitive Behavioral Therapy for BN (CBT-BN; Wilson & Pike, 2001). It is the only empirically supported treatment for BN. This treatment modality targets dysfunctional thoughts and the ensuing behaviors that maintain disordered eating symptomatology. Inherent in this treatment is learning more adaptive affective coping strategies. This further supports the critical role of emotion regulation in binge-purge disorders. On average, CBT-BN eliminates binge eating and purging in about 50% of all patients and reduces bingeing and purging in over 80% of patients (Wilson & Pike, 2001). Focusing on the affective components of treatments may be a key factor in better understanding and managing these often treatment-resistant disorders. Since

affect intensity appears to play an important role in the emotion regulation theory of eating disorders, perhaps teaching patients ways in which to downregulate negative emotions (e.g., mindfulness meditation) and upregulate positive emotions (e.g., relaxation and imagery) without the use of food would be particularly advantageous in this population.

Pharmacology offers additional insight into the ties between eating and emotionality. The antidepressant fluoxetine (a selective serotonin reuptake inhibitor [SSRI]) is the only Federal Drug Administration (FDA) approved medication for the treatment of any eating disorder (U.S. FDA, 1997). In a controlled study examining the effectiveness of fluoxetine for bingeing-purging, episodes decreased by 80% in the fluoxetine group compared to controls (Walsh, et al., 2000). Again, this highlights the importance of mood and emotionality in these disorders based on the 5-HT regulatory function of SSRIs. Unfortunately, in patients with AN, SSRIs have not been shown to produce significant or consistent improvements in terms of either weight gain or giving up one's pursuit of thinness (Krüger & Kennedy, 2000). Further study of medications targeting emotionality may prove beneficial among those with an eating disorder.

Limitations

The present study has important limitations that should be addressed in future research. Limitations of this study include a smaller than desired sample size and retrospective assessment. Evaluation of the restrictor subgroup was particularly underpowered (accounting for 34.9% of the total sample). The use of an inpatient

sample and reliance on nursing staff to collect data made sample size difficult to control.

Because this was a new line of inquiry, some analyses were exploratory in nature. Furthermore, small sample size and the clustering (or interdependence) of variables made overestimation of significance a possible risk and increased the risk of Type I errors (Cohen, Cohen, West, & Aiken, 2003). A more suitable means to analyze the complicated relationship between eating and emotional outcomes would be structural equation modeling (SEM). SEM allows flexible and powerful examination of the relationships between observed and latent variables as well as the ability to test cross-group similarities and differences among multiple latent variables (Kline, 1998). However, based on the obtained sample size, this method was not feasible and other statistical analyses were deemed adequate for a preliminary test of hypotheses generated by the theory presented in this study.

Although the small sample size reduced power to detect relationships, reliance on retrospective recall and cross-sectional data are more serious problems. It is difficult for patients to remember how they felt immediately following a period of disordered eating. Recollections may also be clouded by specific memories (both positive and negative) as well as by the way one thinks they would feel, rather than how they actually felt. To counteract the effects of retrospective recall and test the emotion regulation theory of eating disorders even further, real time (EMA) or in vivo (neuroimaging) technology would be better suited to study the complex and multifaceted relationship of eating and emotions.

Conclusions

Results of the present study support the theory that affective differences exist between individuals who solely restrict dietary intake and those who also engage in binge-purge behaviors. It appears that affect intensity may be one of the most important differences. Binge-purgers had marginally higher levels of affect intensity than did restrictors. More important than group differences, affect intensity moderated the emotional outcomes of a number of disordered eating behaviors in both groups. This construct warrants further study in disordered eating populations.

Based on the chronicity of eating disorders and the paucity of effective treatment modalities, this is an imperative area of research. The present study has shed light on some of the affective influences and outcomes of eating disorder symptomatology and preliminarily validates the emotion regulation theory of eating disorders. Understanding the *functionality* of specific disordered eating behaviors may serve to proliferate a greater understanding of these extremely *dysfunctional* disorders.

REFERENCES

- Abraham, H.D., & Joseph, A.B. (1986). Bulimic vomiting alters pain tolerance and mood. *International Journal of Psychiatry in Medicine*, 16(4), 311-316.
- Aiken, L.S., & West, S.G. (1991). *Multiple regression: Testing and interpreting interactions*. Thousand Oaks: Sage.
- American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders/Text Revision (4th ed.)*. Washington DC: APA.
- American Psychiatric Association (APA). Retrieved May 30, 2010, Posted 2010 at <http://www.dsm5.org/ProposedRevisions/Pages/EatingDisorders.aspx>.
- Barbarich, N.C., McConaha, C.W., Gaskill, J., La Via, M., Frank, G.K., Achenbach, S., Plotnicov, K.H., & Kaye, W.H. (2004). An open trial of olanzapine in anorexia nervosa. *Journal of Clinical Psychiatry*, 65(11), 1480-1482.
- Beck, A.T. (1976). *Cognitive therapy and the emotional disorders*. New York: International Universities Press.
- Beck, A.T., Steer, R.A., & Garbin, M.G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review*, 8(1), 77-100.
- Beck, A.T., Ward, C.H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561-571.

- Blumberg, S.H., & Izard, C.E. (1985). Affective and cognitive characteristics of depression in 10- and 11-year-old children. *Journal of Personality and Social Psychology*, 49(1), 194-202.
- Blundell, J.E. (1984). Serotonin and appetite. *Neuropharmacology*, 23, 1537-1551.
- Booth, D.A. (1994). *Psychology of nutrition*. London: Taylor & Francis.
- Bruch, H. (1973). *Eating disorders: Obesity, anorexia nervosa, and the person within*. New York: Basic Books.
- Buchanan, T.W., Tranel, D., & Adolphs, R. (2006). Memories for emotional autobiographical events following unilateral damage to medial temporal lobe. *Brain*, 129(1), 115-127.
- Bulik, C.M., Berkman, N.D., Brownley, K.A., Sedway, J.A., & Lohr, K.N. (2007). Anorexia nervosa treatment; A systematic review of randomized controlled trials. *International Journal of Eating Disorders*, 40, 310-320.
- Bush, G., Luu, P., & Posner, M.I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Science*, 4, 215-222.
- Bydlowski, S., Corcos, M., Jeammet, P., Paterniti, S., Berthoz, S., Laurier, C., et al. (2005). Emotion-processing deficits in eating disorders. *International Journal of Eating Disorders*, 37, 321-329.
- Carroll, J.M., Touyz, S.W., & Beumont, P.J.V. (1996). Specific comorbidity between bulimia nervosa and personality disorders. *International Journal of Eating Disorders*, 19(2), 159-170.

- Carter, J.D., Joyce, P.R., Mulder, R.T., Luty, S.E., & Sullivan, P.F. (1999). Gender differences in the rate of comorbid axis I disorders in depressed outpatients. *Depression and Anxiety*, 9, 49-53.
- Casper, R.C. (1998). Depression and eating disorders. *Depression and Anxiety*, 8 (Supplement 1), 96-104.
- Cockell, S.J., Hewitt, P.L., Seal, B., Sherry, S., Goldner, E.M., Flett, G.L., et al. (2002). Trait and self-presentational dimensions of perfectionism among women with anorexia nervosa. *Cognitive Therapy and Research*, 26(6), 745-748.
- Cohen, J., Cohen, P., West, S.G., & Aiken, L.S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). New Jersey: Lawrence Erlbaum Associates, Inc.
- Crisp, A.H. (1984). The psychopathology of anorexia nervosa: Getting the "heat" out of the system. In A.J. Stunkard & E. Steller (Eds.), *Eating and its disorders* (pp. 209-234). New York: Raven.
- Davis, C., & Claridge, G. (1998). The eating disorders as addiction: A psychobiological perspective. *Addictive Behaviors*, 23(4), 463-475.
- Derryberry, D., & Rothbart, M.K. (1988). Arousal, affect, and attention as components of temperament. *Journal of Personality and Social Psychology*, 55(6), 958-966.
- Desmet, P.M.A., & Schifferstein, H.N.J. (2008). Sources of positive and negative emotions in food experience. *Appetite*, 50, 290-301.

- Drevets, W.C., Price, J.L., Simpson, J.R. Jr., Todd, R.D., Reich, T., Vannier, M., & Raichle, M.E. (1997). Subgenual prefrontal cortex abnormalities in mood disorders. *Nature*, 386, 824-827.
- Eisler, I., Dare, C., Russell, G.F.M., Szmulker, G.I., LeGrange, D., & Dodge, E. (1997). Family and individual therapy in anorexia nervosa: A five-year follow-up. *Archives of General Psychiatry*, 54, 1025-1030.
- Eysenck, H.J. (1981). General features of the model. In H.J. Eysenck (Ed.), *A model for personality* (pp.1-37). New York: Springer-Verlag.
- Fairburn, C.G. & Cooper, Z. (1993). The eating disorder examination (12th ed.). In C. Fairburn & G. Wilson (Eds.), *Binge eating: Nature, assessment, and treatment* (pp. 317-360). New York: Guilford Press.
- Fava, M., Abraham, M., Clancy-Colecchi, K., Pava, J.A., Matthews, J., & Rosenbaum, J.F. (1997). Eating disorder symptomatology in major depression. *Journal of Nervous and Mental Disorders*, 185, 140-144.
- Frank, G.K., Kaye, W.H., Meltzer, C.C., Price, J.C., Greer, P., McConaha, C., et al. (2002). Reduced 5-HT_{2A} receptor binding after recovery from anorexia nervosa. *Biological Psychiatry*, 52, 896-906.
- Franko, D.L., & Keel, P.K. (2006). Suicidality in eating disorders: Occurrence, correlates, and clinical implications. *Clinical Psychology Review*, 26, 769-782.

- Garner, D.M. (1991). *The Eating Disorders Inventory-2*. Odessa, FL: Psychological Assessment Resources.
- Garner, D.M., Olmstead, M.P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders*, 2, 15-34.
- Goldin, P.R., McRae, K., Ramel, W., & Gross, J.J. (2008). The neural basis of emotion regulation: Reappraisal and suppression of negative emotion. *Biological Psychiatry*, 63, 577-586.
- Gross, J.J. (1998a). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology*, 74(1), 224-237.
- Gross, J.J. (1998b). The emerging field of emotion regulation: An integrative review. *Review of General Psychology*, 2(3), 271-299.
- Gross, J.J. (2001). Emotion regulation in adulthood: Timing is everything. *Current Directions in Psychological Science*, 10(6), 214-219.
- Groth-Marnat, G. (1990). *The Handbook of psychological assessment* (2nd ed.), New York: John Wiley & Sons.
- Hamilton, N., Karoly, P., Gallagher, M., Stevens, N., Karlson, C., & McCurdy, D. (2009). The assessment of emotion regulation in cognitive context: the emotion amplification and reduction scales. *Cognitive Therapy and Research*, 33, 255-263.

- Harrison, A., Sullivan, S., Tchanturia, K., & Treasure, J. (2009). Emotion recognition and regulation in anorexia nervosa. *Clinical Psychology and Psychotherapy*, 16, 348-356.
- Hawkins, R.C., II, & Clement, P.F. (1984). Binge eating: Measurement problems and a conceptual model. In R.C. Hawkins, W.J. Fremouw, & P.F. Clement (Eds.), *The binge purge syndrome: Diagnosis, treatment, and research*. New York: Springer.
- Herman, C.P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality*, 43, 647-660.
- Herman, C.P., & Polivy, J. (1980). Restrained eating. In Stunkard, A.J. (Ed.), *Obesity* (pp. 208-225). Philadelphia: W.B. Saunders.
- Herman, C.P., & Polivy, J. (1988). Studies of eating in normal dieters. In Walsh, B.T. (Ed.), *Eating Behavior in Eating Disorders* (pp. 95-112). Washington D.C.: American Psychiatric Association Press.
- Herman, C.P., Polivy, J., & Esses, V.M. (1987). The illusion of counter-regulation. *Appetite*, 9, 161-169.
- Herzog, D.B., Keller, M.C., Lavori, P.W., Kenny, G.M., & Sacks, N.R. (1992). The prevalence of personality disorders in 210 women with eating disorders. *Journal of Clinical Psychiatry*, 53, 147-152.
- Jeppson, J.E., Richards, P.S., Hardman, R.K., & MacGranley, H. (2003). Binge and purge processes in bulimia nervosa: A qualitative investigation. *Eating Disorders*, 11, 115-128.

- Kaye, W.H. (2008). Neurobiology of anorexia and bulimia nervosa. *Physiology and Behavior*, 94, 121–135.
- Kaye, W.H., Barbarich, N.C., Putnam, K., Gendall, K.A., Fernstrom, J., Fernstrom, M., et al. (2003). Anxiolytic effects of acute tryptophan depletion in anorexia nervosa. *International Journal of Eating Disorders*, 33, 257-267.
- Kaye, W.H., Frank, G.K., Bailer, U.F., & Henry, S.E. (2005). Neurobiology of anorexia nervosa: Clinical implications of alterations of the function of serotonin and other neuronal systems. *International Journal of Eating Disorders*, 37, S15-S19.
- Kaye, W.H., Gendall, K.A., Fernstrom, M.H., Fernstrom, J.D., McConaha, C.W., & Weltzin, T.E. (2000). Effects of acute tryptophan depletion on mood in bulimia nervosa. *Biological Psychiatry*, 47, 151-157.
- Kaye, W.H., Greeno, C.G., Moss, H., Fernstrom, J., Fernstrom, M., Lilenfeld, L.R., et al. (1998). Alterations in serotonin activity and psychiatric symptoms after recovery from bulimia nervosa. *Archives of General Psychiatry*, 55, 927-935.
- Kaye, W.H., & Weltzin, T.E. (1991). Neurochemistry of bulimia nervosa. *Journal of Clinical Psychiatry*, 52(Supplement), 21-28.
- Keel, P.K., & Herzog, D.B. (2004). Long-term outcome, course of illness, and mortality and in anorexia nervosa, bulimia nervosa, and binge eating disorder. In T.D. Brewerton (Ed.) *Clinical handbook of eating disorders: An integrated approach* (pp. 97-116). New York: Informa Health Care.

- Kline, R.B. (1998). *Principles and practice of structural equation modeling*. New York: Guilford Press.
- Krüger, S., & Kennedy, S.H. (2000). Psychopharmacotherapy of anorexia nervosa, bulimia nervosa, and binge-eating disorder. *Journal of Psychiatry and Neuroscience*, 25(5), 497-508.
- Larsen, R.J., & Diener, E. (1984, May). *Cognitive operations associated with the characteristic of emotional response intensity*. Paper presented at the meeting of the Midwestern Psychological Association, Chicago.
- Larsen, R. J., & Diener, E. (1987). Affect intensity as an individual difference characteristic: A review. *Journal of Research in Personality*, 21, 1-39.
- Larsen, R.J., Diener, E., & Emmons, R.A. (1986). Affect intensity and reactions to daily life events. *Journal of Personality and Social Psychology*, 51(4), 803-814.
- Markey, M.A., & Vander Wal, J.S. (2007). The role of emotional intelligence and negative affect in bulimic symptomatology. *Comprehensive Psychiatry*, 48, 458-464.
- Mauss, I. B., Cook, C. L., & Gross, J. J. (2007). Automatic emotion regulation during anger provocation. *Journal of Experimental Social Psychology*, 43, 698-711.
- McCarthy, M. (1990). The thin ideal, depression, and eating disorders in women. *Behavioral Research and Therapy*, 28, 205-218.

- Miyake, Y., Okamoto, Y., Onoda, K., Shirao, N., Okamoto, Y., Otagaki, Y., & Yamawaki, S. (2010). Neural processing of negative word stimuli concerning body image in patients with eating disorders: An fMRI study. *NeuroImage*, 50, 1333-1339.
- O'Brien, K.M., & Vincent, N.K. (2003). Psychiatric comorbidity in anorexia and bulimia nervosa: Nature, prevalence, and causal relationships. (2003). *Clinical Psychology Review*, 23, 57-74.
- Ochsner, K.N. (2007). How thinking controls feeling: A social cognitive neuroscience approach. In Harmon-Jones & Winkielman (Eds.), *Social neuroscience: Integrating biological and psychological explanations of social behavior* (pp. 106-133). New York: Guilford Press.
- Overton, A., Selway, S., Strongman, K., & Houston, M. (2005). Eating disorders – The regulation of positive as well as negative emotion experience. *Journal of Clinical Psychology in Medical Settings*, 12(1), 39-56.
- Preacher, K.J., Curran, P.J., & Bauer, D.J. (2006). Computational tools for probing interaction effects in multiple linear regression, multilevel modeling, and latent curve analysis. *Journal of Educational and Behavioral Statistics*, 31, 437-448.
- Rome, E.S., & Ammerman, S. (2003). Medical complications of eating disorders: An update. *Journal of Adolescent Health*, 33, 418-426.

- Rosenvinge, J.H., Martinussen, M., & Ostensen, E. (2000). The comorbidity of eating disorders and personality disorders: A meta-analytic review of studies published between 1983 and 1998. *Eating and Weight Disorders*, 5, 52-61.
- Russell, G.F., Szmukler, G.I., Dare, C., & Eisler, I. (1987). An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry*, 44(12), 1047-1056.
- Safer, D.L., Telch, C.F., & Agras, W.S. (2001). Dialectical behavior therapy adapted for bulimia: A case report. *International Journal of Eating Disorders*, 30, 101-106.
- Santonastaso, P., Friederici, S., & Favaro, A. (1999). Full and partial syndromes in eating disorders: A 1-year prospective study of risk factors among female students. *Psychopathology*, 32, 50-56.
- Sexton, M.C., Sunday, S.R., Hurt, S., & Halmi, K.A. (1998). The relationship between alexithymia, depression, and axis II psychopathology in eating disorder inpatients. *International Journal of Eating Disorders*, 23, 277-286.
- Siegle, G.J. (2007). Brain mechanisms of borderline personality disorder at the intersection of cognition, emotion, and the clinic. *American Journal of Psychiatry*, 164(12), 1776-1779.
- Sim, L., & Zeman, J. (2006). The contribution of emotion regulation to body dissatisfaction and disordered eating in early adolescent girls. *Journal of Youth and Adolescence*, 35(2), 219-228.

- Steiger, H. (2004). Eating disorders and the serotonin connection: state, trait and developmental effects. *Journal of Psychiatry and Neuroscience*, 29(1), 20-29.
- Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin*, 128(5), 825-848.
- Stice, E., Fischer, M., & Martinez, E. (2004). Eating disorder diagnostic scale: Additional evidence of reliability and validity. *Psychological Assessment*, 16(1), 60-71.
- Stice, E., Nemeroff, C., & Shaw, H.E. (1996). Test of the dual pathway model of bulimia nervosa: Evidence for dietary restraint and affect regulation mechanisms. *Journal of Social and Clinical Psychology*, 15(3), 340-363.
- Stice, E., Telch, C.F., & Rizvi, S.L. (2000). Development and validation of the eating disorder diagnostic scale: A brief self-report measure of anorexia, bulimia, and binge-eating disorder. *Psychological Assessment*, 12(2), 123-131.
- Striegel-Moore, R.H., Silberstein, L.R., & Rodin, J. (1986). Toward an understanding of risk factors for bulimia. *American Psychologist*, 41, 246-263.
- Strober, M., Freeman, R., & Morrell, W. (1997). The long term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse and outcome predictors over 10-15 years in a prospective study. *International Journal of Eating Disorders*, 22, 339-360.
- Stokes, P.E. & Sikes, C.R. (1991). Hypothalamic-pituitary-adrenal axis in psychiatric disorders. *Annual Review of Medicine*, 42, 519-531.

- Sullivan, P.F. (1995). Mortality in anorexia nervosa. *Archives of General Psychiatry*, 152, 1073-1074.
- Sullivan, P.F. (2002). Course and outcome of anorexia nervosa and bulimia nervosa. In C.G. Fairburn & K.D. Brownell (Eds.), *Eating disorders and obesity: A comprehensive handbook, 2nd edition*. (pp. 226-232). New York: Guilford Press.
- Swinbourne, J.M. & Touyz, S.W. (2007). The co-morbidity of eating disorders and anxiety disorders: a review. *European Eating Disorders Review*, 15, 253-274.
- Sylvester, C.J., & Forman, S.F. (2008). Clinical practice guidelines for treating restrictive eating disorder patients during medical hospitalization. *Current Opinion in Pediatrics*, 20(4), 390-397.
- Telch, C.F., Safer, W.S., & Linehan, M.M. (2001). Dialectical behavior therapy for binge eating disorder. *Journal of Consulting and Clinical Psychology*, 69(6), 1061-1065.
- Thompson-Brenner, H., Glass, S., & Westen, D. (2003). A multidimensional meta-analysis of psychotherapy for bulimia nervosa. *Clinical Psychology: Science and Practice*, 10(3), 269-287.
- Tiihonen, J., Keski-Rahkonen, A., Loppinen, M., et al. (2004). Brain serotonin 1A receptor binding in bulimia nervosa. *Biological Psychiatry*, 55, 871-873.
- Treasure, J., Claudino, A.M. & Zucker, N. (2010). Eating disorders. *Lancet*, 375, 583-593.

- Turner, H.M., Bryant-Waugh, R., & Peveler, R. (2009). An approach to sub-grouping the eating disorder population: Adding attachment and coping style. *European Eating Disorders Review*, 17(4), 269–280.
- United States Food and Drug Administration (FDA). Retrieved December 2, 2007, Posted: 1/29/1997 at <http://www.fda.gov/medwatch/safety/label/nov96.htm>.
- Van Strien, T. (2000). Ice-cream consumption, tendency toward overeating, and personality. *International Journal of Eating Disorders*, 28, 460-464.
- Vitousek, K., & Ewald, I. (1993). Self representation in eating disorders: A cognitive perspective. In Z. Segal & S. Blatt (Eds.), *The self in emotional disorders* (pp. 221-257). New York: Guilford Press.
- Vitousek, K., & Manke, F. (1994). Personality variables and disorders in anorexia nervosa and bulimia nervosa. *Journal of Abnormal Psychology*, 103(1), 137-147.
- Waller, G., Babbs, M., Milligan, R., Meyer, C., Ohanian, V., & Leung, N. (2003). Anger and core beliefs in the eating disorders. *International Journal of Eating Disorders*, 34, 118-124.
- Walsh, B.T., Agras, W.S., Devlin, M.J., Fairburn, C.G., Wilson, G.T., Kahn, C., et al. (2000). Fluoxetine for bulimia nervosa following poor response to psychotherapy. *American Journal of Psychiatry*, 157(8), 1332-1334.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, 54(6), 1063-1070.

- Wegner, K.E., Smyth, J.M., Crosby, R.D., Wittrock, D., Wonderlich, S.A., & Mitchell, J.E. (2002). An evaluation of the relationship between mood and binge eating in the natural environment using ecological momentary assessment. *International Journal of Eating Disorders*, 32, 352-361.
- Westen, D., & Harnden-Fischer, J. (2001). Personality profiles in eating disorders: Rethinking the distinction between axis I and axis II. *American Journal of Psychiatry*, 158(4), 547-562.
- Williamson, D.A., Gleaves, D.H., & Stewart, T.M. (2005). Categorical versus dimensional model of eating disorders: An examination of the evidence. *International Journal of Eating Disorders*, 37, 1-10.
- Wills, T.A. (1981). Downward comparison principles in social psychology, *Psychological Bulletin*, 90, 245-271.
- Wilson, G.T., & Pike, K.M. (2001). Eating disorders. In *Clinical handbook of psychological disorders*, 3rd edition (pp. 332-375). New York: Guilford Press.
- Zanarini, M.C., Frankenburg, F.R., Dubo, E.D., Sickel, A.E., Trikha, A., Levin, A., et al. (1998). Axis I comorbidity of borderline personality disorder. *American Journal of Psychiatry*, 155, 1733-1739.

TABLES

Table 1. Descriptive characteristics.

	<u>Total Sample</u> (N = 63)	<u>Restrictors</u> (n = 22)	<u>Binge-Purgers</u> (n = 41)
<u>Age</u>	$M = 31.9, SD = 11.77$	$M = 34.6, SD = 11.53$	$M = 30.5, SD = 11.79$
<u>BMI</u>	$M = 20.2, SD = 7.62$	$M = 15.8, SD = 1.96$	$M = 22.5, SD = 8.47$
<u>Ethnicity</u>	95.2% Caucasian	95.5% Caucasian	95.1% Caucasian
<u>Education</u>	23.8% Bachelor's 22.2% High school	27.3% Bachelor's 27.3% High school	22.0% Bachelor's 19.5% High school
<u>Employment</u>	38.1% Full-time 25.4% Unemployed	36.4% Unemployed 27.3% Full-time	43.9% Full-time 19.5% Unemployed
<u>Income</u>	19.0% >\$100,000 14.3% \$10-19,999	27.3% <\$10,000 2.7% >\$100,000	19.5% \$10-19,999 17.1% >\$100,000

Table 2. Correlation matrix.

	Beh. Diag	DSM Diag.	BMI	Age	PA week	NA week	PA restrict	NA restrict	PA binge	NA binge	PA purge	NA purge	TEARS Amp.	TEAR S Reduce	BDI	AIM	Restric t Freq.	Binge Freq.	Purge Freq.
Behavioral Diagnosis	1	.673**	.419**	-.165	.133	.104	-.131	.071	-.231	-.074	-.388**	.037	-.151	.033	.049	.241	.012	.643**	.540**
DSM-IV Diagnosis		1	.622**	-.256*	.082	.023	-.296*	.179	-.159	.048	-.252	.050	-.155	.092	-.045	.163	-.083	.313*	.305*
BMI			1	.008	.190	-.029	-.239	.111	-.138	-.133	-.114	-.040	-.041	.009	-.117	.194	-.189	.166	.019
Age				1	-.124	-.426**	-.046	-.283*	.181	-.402**	-.028	-.129	-.285*	-.049	-.067	-.090	-.008	-.073	-.056
PA week					1	-.090	.133	.321*	.458**	-.262	.149	.199	.233	.303*	-.477**	.340*	-.025	.131	-.006
NA week						1	.333*	.133	-.023	.656**	.180	.288*	.320*	-.128	.525**	.350*	-.044	.109	.076
PA restrict							1	-.450**	.368*	.160	.609**	.024	.386**	-.112	-.112	.035	.056	.025	-.082
NA restrict								1	-.031	.093	-.175	.263	-.014	.138	.180	.246	.098	-.212	.065
PA binge									1	-.272	.500**	.280	.435**	.400**	-.277	.011	.272	-.055	.054
NA binge										1	.070	.293*	.289*	-.351*	.398**	.269	.012	-.097	-.061
PA purge											1	-.251	.509**	-.129	-.169	-.010	.041	-.391**	-.475**
NA purge												1	.116	.081	.147	.271*	.038	.172	.173
TEARS Amplify													1	.090	-.030	.195	.208	-.096	-.212
TEARS Reduce														1	-.132	-.262*	.103	-.042	.156
BDI															1	-.014	.153	-.059	.130
AIM																1	-.002	.342**	.101
Restrict Frequency																	1	-.190	.230
Binge Frequency																		1	.596**
Purge Frequency																			1

** $p < .01$.

* $p < .05$.

Table 3. ANCOVA: behavioral versus diagnostic profiles.

	<u>Behavioral</u>		<u>DSM-IV</u>	
	Restrictor	Binge-Purger	AN	BN
Variable	<i>M</i> ± <i>SD</i> , <i>p</i>	<i>M</i> ± <i>SD</i>	<i>M</i> ± <i>SD</i> , <i>p</i>	<i>M</i> ± <i>SD</i>
Affect Intensity	136.95±21.08, .060*	148.54±23.72	141.09±25.84, .213	148.48±19.70
Depressive Symptomatology	30.41±16.07, .679	31.93±12.45	31.88±14.34, .764	30.83±13.16
Weekly NA**	32.82±9.58, .748	34.78±8.23	33.85±9.29, .482	34.38±8.10
Weekly PA	25.80±9.03, .281	28.34±8.77	26.72±9.10, .483	28.31±8.67
* <i>p</i> < .10 (marginal significance).				
** Age used as covariate.				

Table 4. Independent samples t-test: Restrictors versus Binge-Purgers.

	<u>Restrictors</u> <i>M</i> ± <i>SD</i>	<u>Binge-Purgers</u> <i>M</i> ± <i>SD</i>	<u>t-test</u>
TEARS-Amplify	20.59±5.93	18.83±5.43	.240
TEARS-Reduce	19.41±5.30	19.83±6.53	.799
BDI	30.41±16.07	31.83±12.59	.703
AIM	136.95±21.08	148.70±24.00	.059*
PA week	25.80±9.03	28.25±8.86	.304
NA week	32.82±9.58	34.70±8.32	.423
PA restrict	30.41±10.68	27.64±10.16	.331
NA restrict	22.77±9.27	24.23±10.58	.598
PA binge	22.57±10.01	17.15±8.11	.123
NA binge	42.64±11.14	40.97±7.77	.627
PA purge	34.76±9.22	25.00±11.14	.003**
NA purge	30.26±11.43	31.20±11.73	.782
** $p < .05$.			
* $p < .10$ (marginal significance).			

Table 5. Restrictor correlation matrix.

	Felt Fat	Fat Fear	Weight Judge	Shape Judge	Restrict Freq.	BDI	AIM	PA week	NA week	PA restrict	NA restrict	TEARS Amplify	TEARS Reduce
Felt Fat	1	.801**	.742**	.728**	.334	.417	.415	-.083	.425*	.403	.159	.231	-.401
Fat Fear		1	.645**	.625**	.397	.447*	.397	-.146	.627**	.416	.117	.195	-.331
Weight Judge			1	.992**	.127	.343	.334	-.123	.386	.294	.089	.160	-.530*
Shape Judge				1	.145	.344	.310	-.151	.353	.299	.028	.123	-.546**
Restrict Freq.					1	.511*	.325	-.312	.221	.080	.054	.135	-.049
BDI						1	.167	-.736**	.563**	.036	.096	.144	-.290
AIM							1	.199	.497*	.334	.558**	.513*	-.228
PA week								1	-.210	.321	.160	.246	.241
NA week									1	.490*	.137	.441*	-.225
PA restrict										1	-.227	.387	-.254
NA restrict											1	.290	-.080
TEARS-Amplify												1	-.116
TEARS-Reduce													1

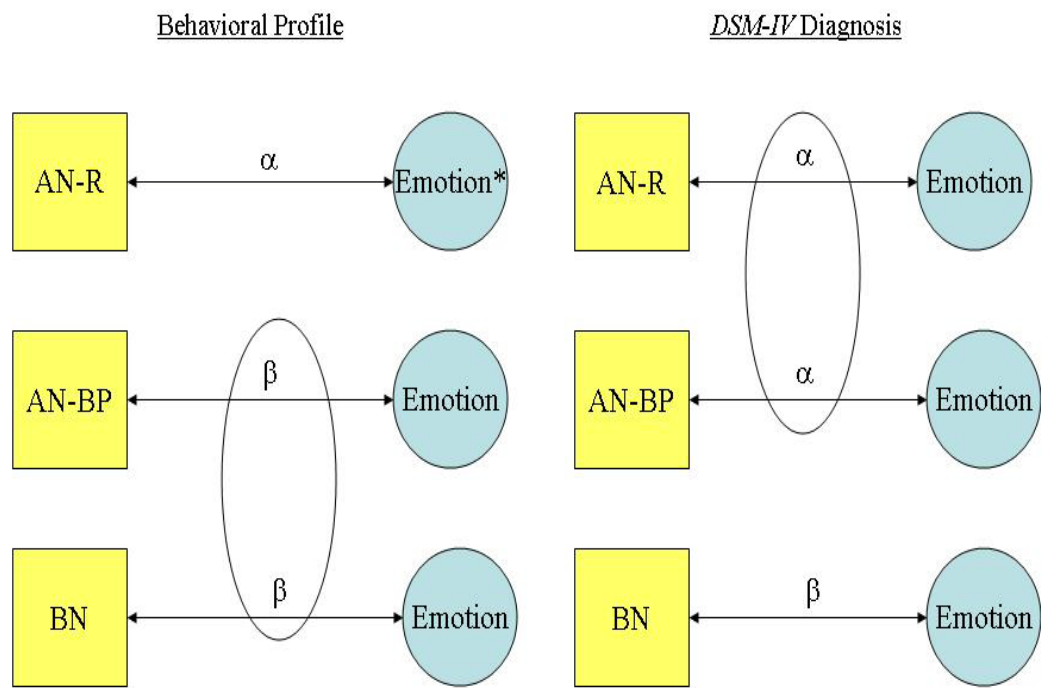
** $p < .01$.

* $p < .05$.

Table 6. Binge-purger correlation matrix.

	Weight Judge	Shape Judge	Binge Freq.	Vomit Freq.	Lax. Freq.	Restrict Freq.	Exer. Freq.	BDI	AIM	PA week	NA week	PA restrict	NA restrict	PA binge	NA binge	PA purge	NA purge	TEARS Amplify	TEARS Reduce
Weight Judge	1	.827**	-.304	-.322*	.168	.240	.407**	.050	-.019	-.001	.255	.103	-.053	-.007	.129	.309	.012	.311*	-.148
Shape Judge		1	-.200	-.268	.125	.096	.244	.068	.039	-.007	.319*	.141	-.080	.003	.107	.163	.142	.166	-.213
Binge Freq.			1	.520**	-.014	-.329*	-.226	-.081	.241	-.014	.121	.107	-.342*	.030	.000	-.246	.204	-.072	-.129
Vomit Freq.				1	.125	.137	.052	-.152	-.054	.104	.016	.104	.010	.186	-.027	-.270	.139	-.235	.243
Lax. Freq.					1	.347*	.578**	-.007	-.192	.167	-.038	-.018	.116	.316*	-.053	.193	-.049	.366*	.196
Restrict Freq.						1	.352*	-.076	-.237	.101	-.196	.014	.160	.223	.147	.083	-.056	.239	.129
Exer. Freq.							1	.133	-.183	.017	.362*	.277	.146	.229	.297	.462**	.049	.420**	.166
BDI								1	-.145	-.322*	.492**	-.230	.240	-.270	.388*	-.171	.257	-.146	-.052
AIM									1	.377*	.253	-.068	.086	.028	.266	.002	.200	.099	-.299
PA week										1	-.042	.050	.400*	.460**	-.241	.139	.153	.263	.330*
NA week											1	.239	.124	.024	.641**	.203	.366*	.273	-.089
PA restrict												1	-.572**	.380*	.204	.459**	.081	.363*	-.040
NA restrict													1	-.072	.017	-.204	.110	-.172	.230
PA binge														1	-.201	.501**	.164	.530**	.429**
NA binge															1	.126	.377*	.148	-.375*
PA purge																1	-.333*	.576**	-.037
NA purge																	1	.025	.051
TEARS-Amp																		1	.197
TEARS-Red																			1
** $p < .01$																			
* $p < .05$.																			

FIGURES



*Emotion Dysregulation: BDI, AIM, and PANAS

Figure 1. Behavioral versus diagnostic profiles.

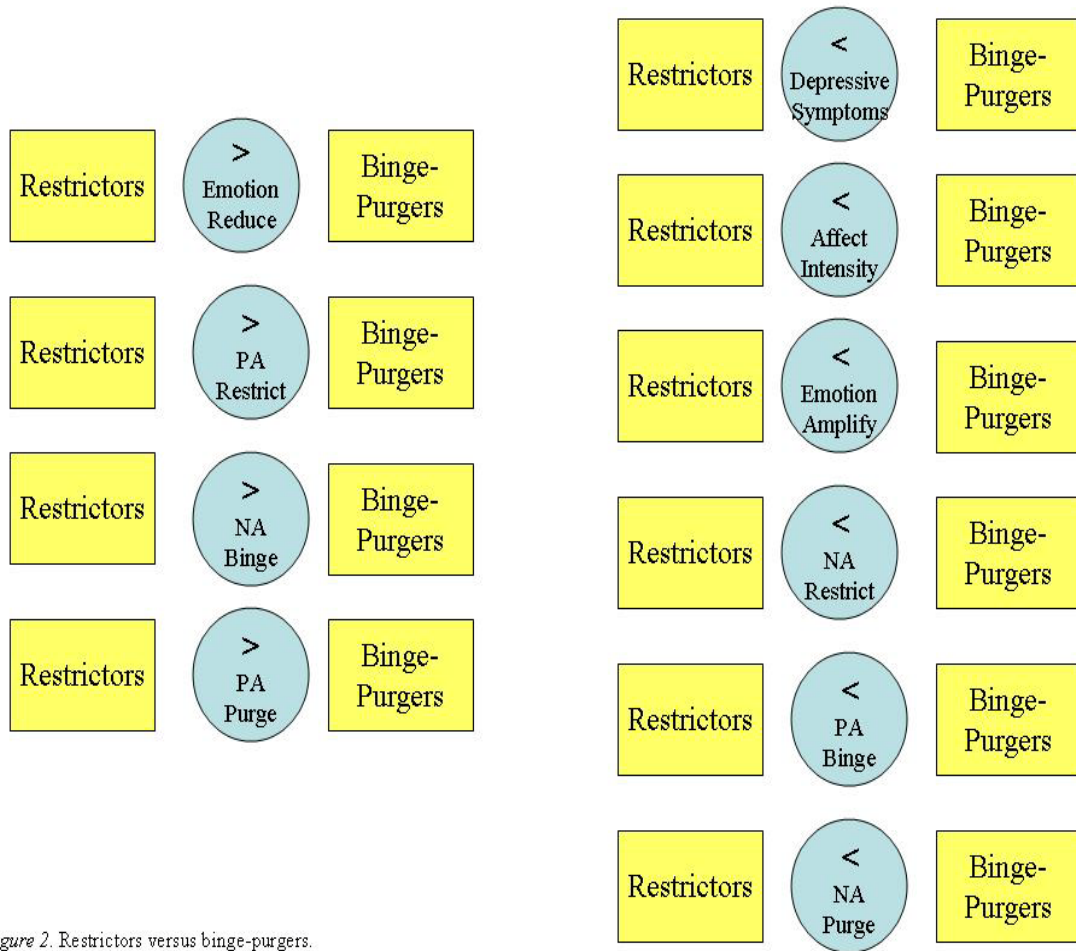


Figure 2. Restrictors versus binge-purgers.

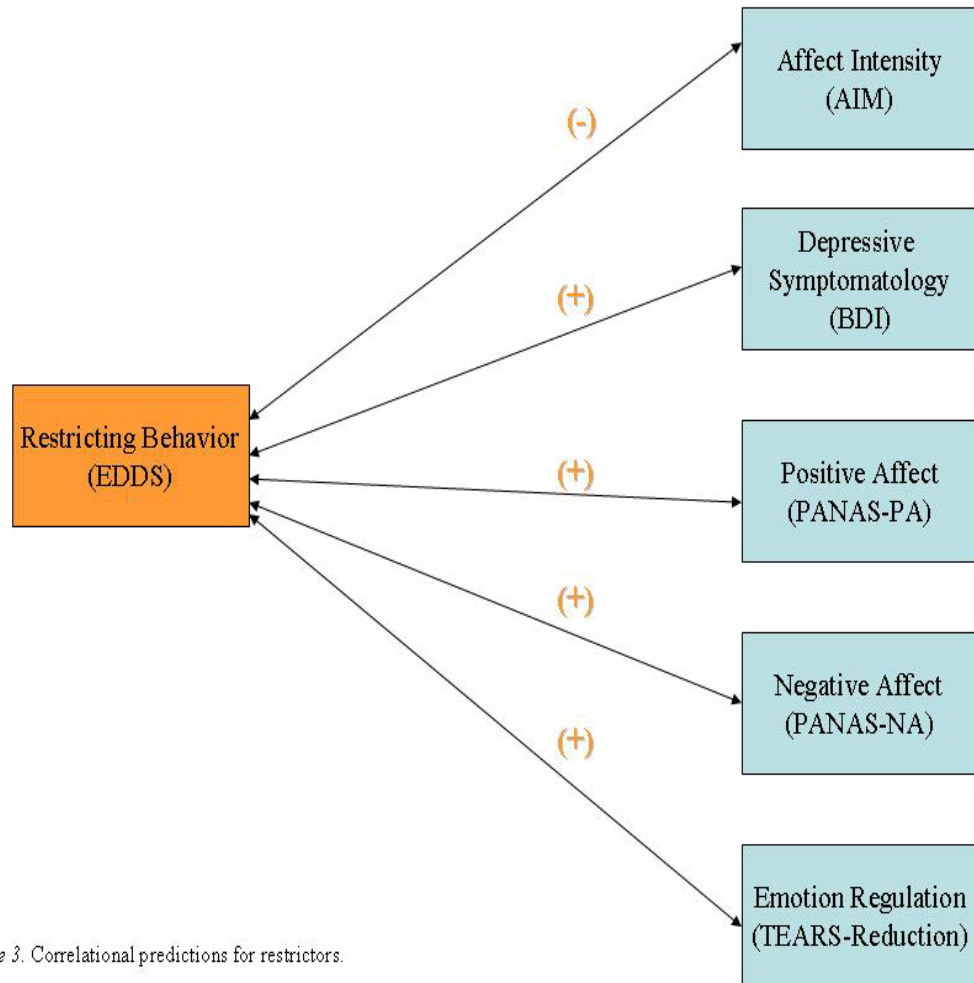


Figure 3. Correlational predictions for restrictors.

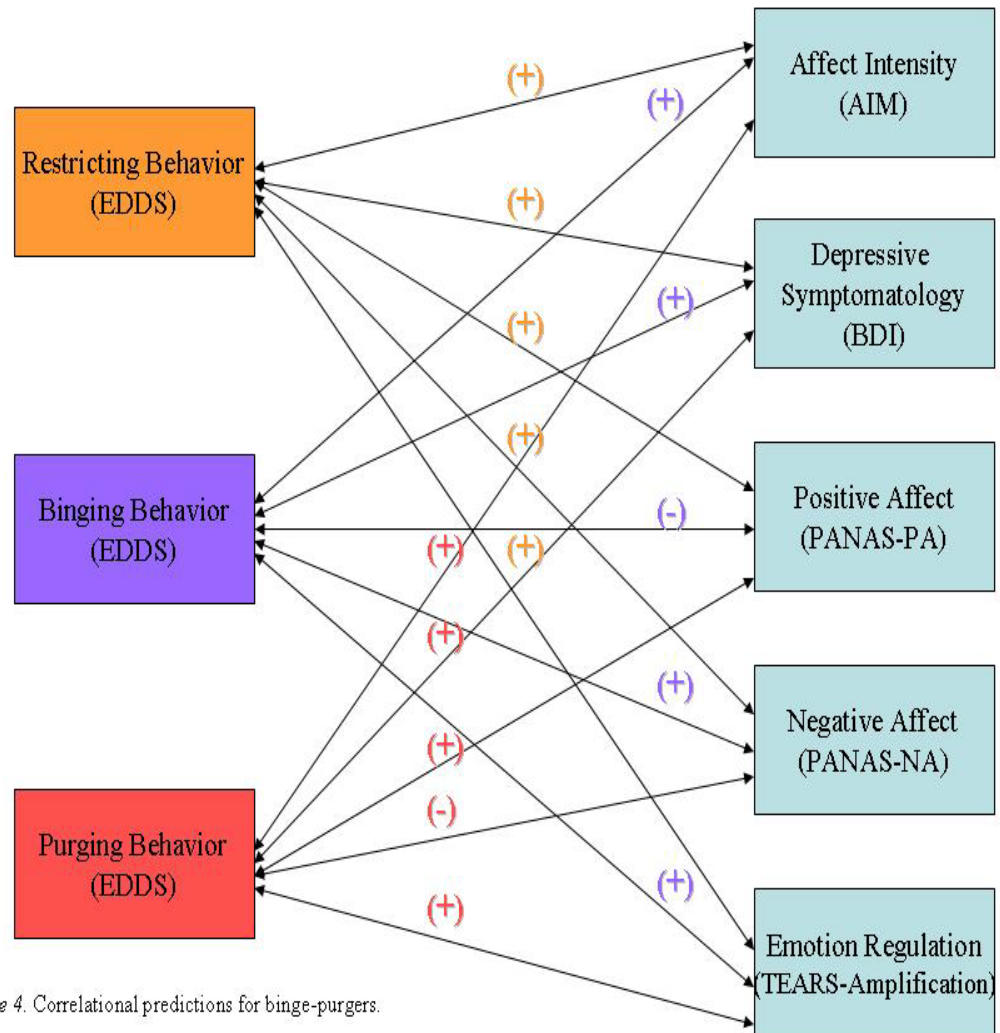


Figure 4. Correlational predictions for binge-purgers.

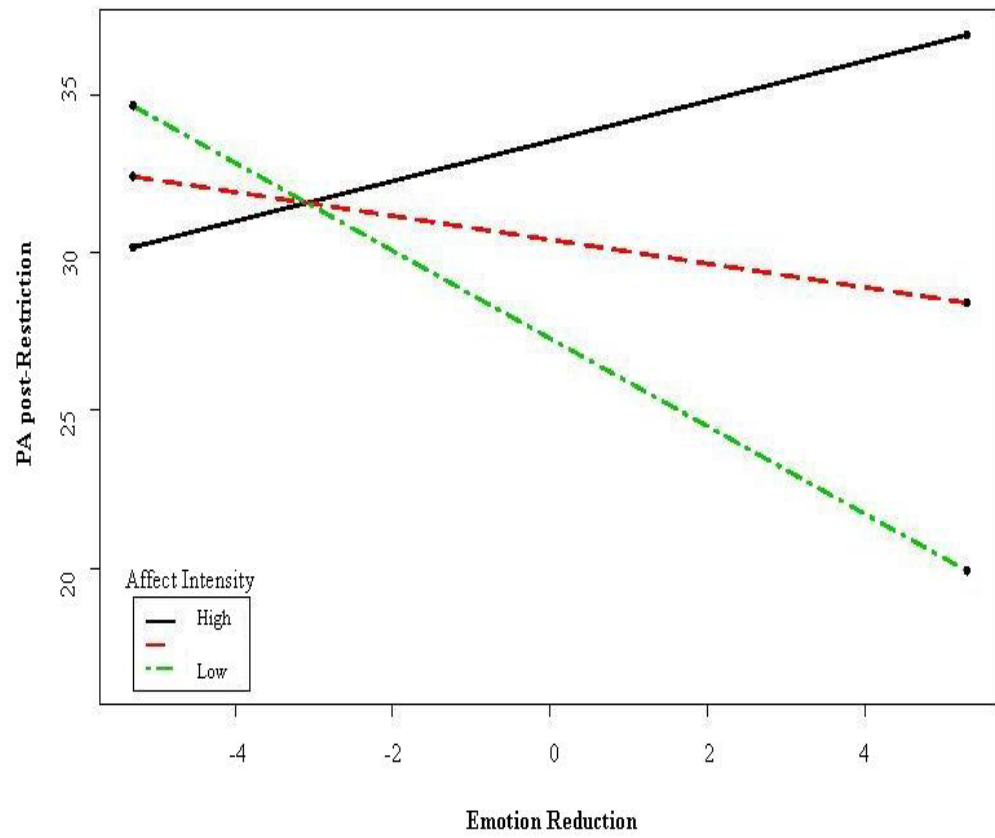


Figure 5. Restrictors interaction: emotion reduction x affect intensity.

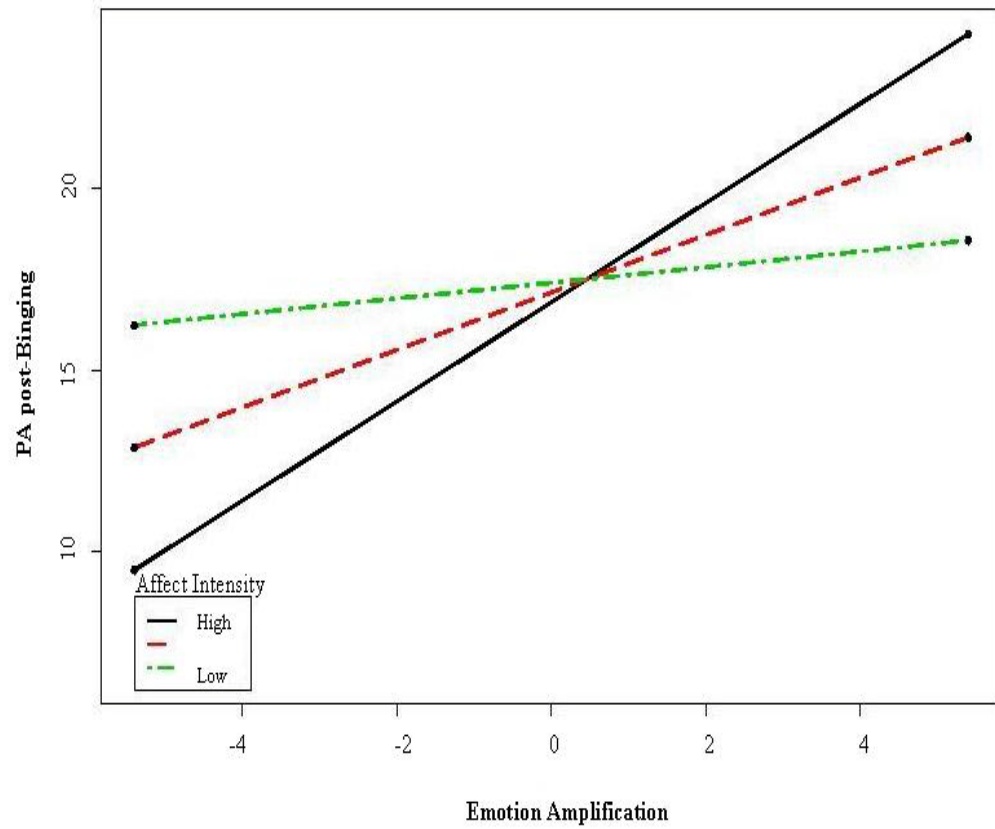


Figure 6. Binge-purgers interaction: emotion amplification x affect intensity.

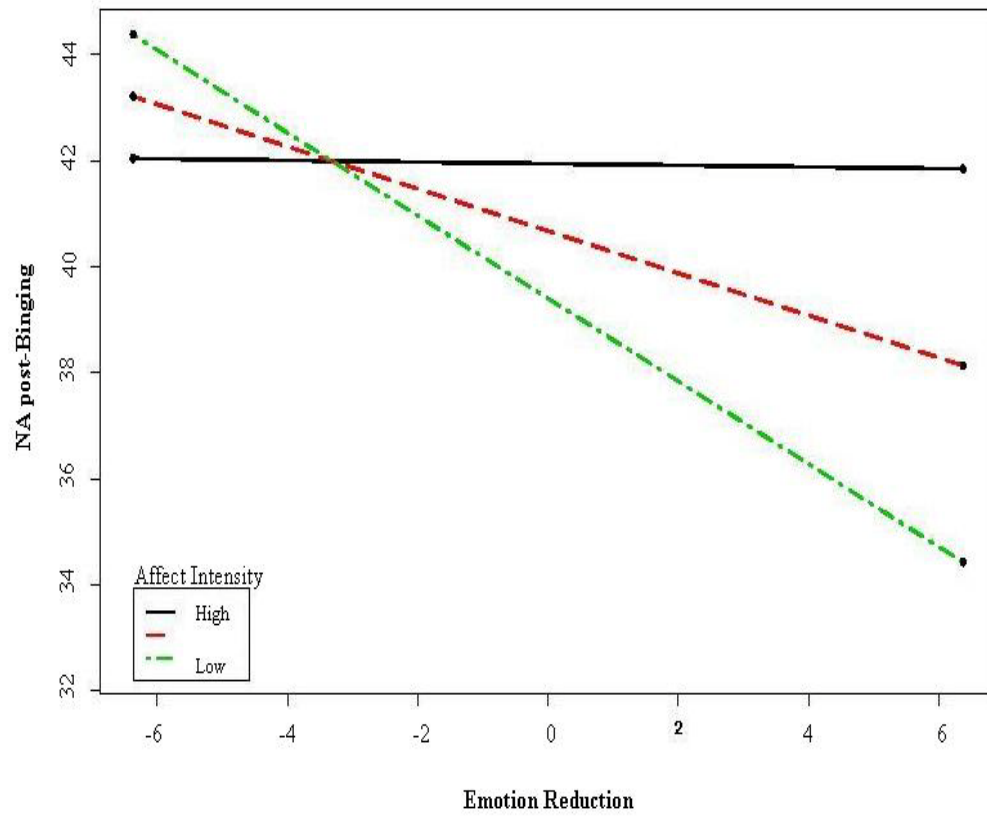


Figure 7. Binge-purgers interaction: emotion reduction x affect intensity.

APPENDIX A: Eating Disorder Diagnostic Scale

Please carefully complete all questions.

Over the past 3 months...	Not at all	Slightly	Moderately	Extremely											
1. Have you felt fat?	0	1	2	3	4	5	6								
2. Have you had a definite fear that you might gain weight or become fat?	0	1	2	3	4	5	6								
3. Has your weight influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6								
4. Has your shape influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6								
5. During the past 6 months have there been times when you felt you have eaten what other people would regard as an unusually large amount of food (e.g., a quart of ice cream) given the circumstances? YES NO															
6. During the times when you ate an unusually large amount of food, did you experience a loss of control (feel you couldn't stop eating or control what or how much you were eating)? YES NO															
7. How many DAYS per week on average over the past 6 MONTHS have you eaten an unusually large amount of food and experienced a loss of control?	0	1	2	3	4	5	6	7							
8. How many TIMES per week on average over the past 3 MONTHS have you eaten an unusually large amount of food and experienced a loss of control?	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14

During these episodes of overeating and loss of control did you...

9. Eat much more rapidly than normal? YES NO
10. Eat until you felt uncomfortably full? YES NO
11. Eat large amounts of food when you didn't feel physically hungry? YES NO
12. Eat alone because you were embarrassed by how much you were eating? YES NO
13. Feel disgusted with yourself, depressed, or very guilty after overeating? YES NO
14. Feel very upset about your uncontrollable overeating or resulting weight gain? YES NO
15. How many times per week on average over the past 3 months have you made yourself vomit to prevent weight gain or counteract the effects of eating? 0 1 2 3 4 5 6 7 8 9 10 11 12 13 14
16. How many times per week on average over the past 3 months have you used laxatives or diuretics to prevent weight gain or counteract the effects of eating?
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14
17. How many times per week on average over the past 3 months have you fasted (skipped at least 2 meals in a row) to prevent weight gain or counteract the effects of eating?
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14
18. How many times per week on average over the past 3 months have you engaged in excessive exercise specifically to counteract the effects of overeating episodes?
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14
19. How much do you weigh? If uncertain, please give your best estimate. ____lb
20. How tall are you? ____ft ____in.
21. Over the past 3 months, how many menstrual periods have you missed? 1 2 3 4 na
22. Have you been taking birth control pills during the past 3 months? YES NO

From Stice, E., Telch, C.F., & Rizvi, S.L. (2000). Development and validation of the eating disorder diagnostic scale: A brief self-report measure of anorexia, bulimia, and binge-eating disorder. *Psychological Assessment*, 12(2), 123-131.

APPENDIX B: Affect Intensity Measure

DIRECTIONS: The following questions refer to the emotional reaction to typical life-events. Please indicate how YOU react to these events by placing a number from the following scale in the blank space preceding each item. Please base your answers on how YOU react, *not* on how you think others react or how you think a person should react.

- | | ALMOST
NEVER | NEVER | OCCASIONALLY | USUALLY | ALMOST
ALWAYS | ALWAYS |
|--|-----------------|-------|--------------|---------|------------------|--------|
| | 1 | 2 | 3 | 4 | 5 | 6 |
1. _____ When I accomplish something difficult I feel delighted or elated.
 2. _____ When I feel happy it is a strong type of exuberance.
 3. _____ I enjoy being with other people very much.
 4. _____ I feel pretty bad when I tell a lie.
 5. _____ When I solve a small personal problem, I feel euphoric.
 6. _____ My emotions tend to be more intense than those of most people.
 7. _____ My happy moods are so strong that I feel like I'm "in heaven."
 8. _____ I get overly enthusiastic.
 9. _____ If I complete a task I thought was impossible, I am ecstatic.
 10. _____ My heart races at the anticipation of some exciting event.
 11. _____ Sad movies deeply touch me.
 12. _____ When I'm happy it's a feeling of being untroubled and content rather than being zestful and aroused.
 13. _____ When I talk in front of a group for the first time my voice gets shaky and my heart races.
 14. _____ When something good happens, I am usually much more jubilant than others.
 15. _____ My friends might say I'm emotional.
 16. _____ The memories I like the most are of those of times when I felt content and peaceful rather than zestful and enthusiastic.
 17. _____ The sight of someone who is hurt badly affects me strongly.
 18. _____ When I'm feeling well it's easy for me to go from being in a good mood to being really joyful.
 19. _____ "Calm and cool" could easily describe me.
 20. _____ When I'm happy I feel like I'm bursting with joy.
 21. _____ Seeing a picture of some violent car accident in a newspaper makes me feel sick to my stomach.
 22. _____ When I'm happy I feel very energetic.
 23. _____ When I receive an award I become overjoyed.
 24. _____ When I succeed at something, my reaction is calm contentment.
 25. _____ When I do something wrong I have strong feelings of shame and guilt.
 26. _____ I can remain calm even on the most trying days.

- 27. _____ When things are going good I feel “on top of the world.”
- 28. _____ When I get angry it’s easy for me to still be rational and not overreact.
- 29. _____ When I know I have done something very well, I feel relaxed and content rather than excited and elated.
- 30. _____ When I do feel anxiety it is normally very strong.
- 31. _____ My negative moods are mild in intensity.
- 32. _____ When I am excited over something I want to share my feelings with everyone.
- 33. _____ When I feel happiness, it is a quiet type of contentment.
- 34. _____ My friends would probably say I’m a tense or “high-strung” person.
- 35. _____ When I’m happy I bubble over with energy.
- 36. _____ When I feel guilty, this emotion is quite strong.
- 37. _____ I would characterize my happy moods as closer to contentment than to joy.
- 38. _____ When someone compliments me, I get so happy I could “burst.”
- 39. _____ When I am nervous I get shaky all over.
- 40. _____ When I am happy the feeling is more like contentment and inner calm than one of exhilaration and excitement.

From Larsen, R. J., & Diener, E. (1987). Affect intensity as an individual difference characteristic: A review. *Journal of Research in Personality*, 21, 1-39.

Scoring: Sum items 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12*, 13, 14, 15, 16*, 17, 18, 19*, 20, 21, 22, 23, 24*, 25, 26*, 27, 28*, 29*, 30, 31*, 32, 33*, 34, 35, 36, 37*, 38, 39, 40*.

* Item is reverse scored.

APPENDIX C: Positive and Negative Affect Schedule – Modified to
Assess Eating Behaviors

PANAS-W

The following words describe different feelings and emotions. Please indicate the extent to which you have felt this way IN THE PAST WEEK. Circle the number that corresponds to your answer choice.

IN THE PAST WEEK, HOW MUCH HAVE YOU FELT. . .		1= VERY SLIGHTLY OR NOT AT ALL 2= A LITTLE 3=MODERATELY 4= QUITE A BIT 5=EXTREMELY	
1. interested	1 2 3 4 5	11. irritable	1 2 3 4 5
2. distressed	1 2 3 4 5	12. alert	1 2 3 4 5
3. excited	1 2 3 4 5	13. ashamed	1 2 3 4 5
4. upset	1 2 3 4 5	14. inspired	1 2 3 4 5
5. strong	1 2 3 4 5	15. nervous	1 2 3 4 5
6. guilty	1 2 3 4 5	16. determined	1 2 3 4 5
7. scared	1 2 3 4 5	17. attentive	1 2 3 4 5
8. hostile	1 2 3 4 5	18. jittery	1 2 3 4 5
9. enthusiastic	1 2 3 4 5	19. active	1 2 3 4 5
10. proud	1 2 3 4 5	20. afraid	1 2 3 4 5

PANAS-R

The following words describe different feelings and emotions. *If applicable*, please indicate the extent to which you have felt this way AFTER you have eaten far less than an average person in one day. Circle the number that corresponds to your answer choice.

HOW MUCH HAVE YOU FELT. . .		1= VERY SLIGHTLY OR NOT AT ALL 2= A LITTLE 3=MODERATELY 4= QUITE A BIT 5=EXTREMELY	
1. interested	1 2 3 4 5	11. irritable	1 2 3 4 5
2. distressed	1 2 3 4 5	12. alert	1 2 3 4 5
3. excited	1 2 3 4 5	13. ashamed	1 2 3 4 5
4. upset	1 2 3 4 5	14. inspired	1 2 3 4 5
5. strong	1 2 3 4 5	15. nervous	1 2 3 4 5
6. guilty	1 2 3 4 5	16. determined	1 2 3 4 5
7. scared	1 2 3 4 5	17. attentive	1 2 3 4 5
8. hostile	1 2 3 4 5	18. jittery	1 2 3 4 5
9. enthusiastic	1 2 3 4 5	19. active	1 2 3 4 5
10. proud	1 2 3 4 5	20. afraid	1 2 3 4 5

PANAS-B

The following words describe different feelings and emotions. *If applicable*, please indicate the extent to which you have felt this way AFTER you have eaten what other people would regard as an unusually large amount of food given the circumstances. Circle the number that corresponds to your answer choice.

HOW MUCH HAVE YOU FELT. . .		1= VERY SLIGHTLY OR NOT AT ALL 2= A LITTLE 3=MODERATELY 4= QUITE A BIT 5=EXTREMELY	
1. interested	1 2 3 4 5	11. irritable	1 2 3 4 5
2. distressed	1 2 3 4 5	12. alert	1 2 3 4 5
3. excited	1 2 3 4 5	13. ashamed	1 2 3 4 5
4. upset	1 2 3 4 5	14. inspired	1 2 3 4 5
5. strong	1 2 3 4 5	15. nervous	1 2 3 4 5
6. guilty	1 2 3 4 5	16. determined	1 2 3 4 5
7. scared	1 2 3 4 5	17. attentive	1 2 3 4 5
8. hostile	1 2 3 4 5	18. jittery	1 2 3 4 5
9. enthusiastic	1 2 3 4 5	19. active	1 2 3 4 5
10. proud	1 2 3 4 5	20. afraid	1 2 3 4 5

PANAS-P

The following words describe different feelings and emotions. *If applicable*, please indicate the extent to which you have felt this way AFTER you have done any of the following behaviors: vomiting to prevent weight gain or counter the effects of overeating, used laxatives or diuretics to prevent weight gain or counter the effects of overeating, skipped at least two meals to prevent weight gain or counter the effects of overeating, and/or engaged in excessive exercise specifically to prevent weight gain or counter the effects of overeating. Circle the number that corresponds to your answer choice.

HOW MUCH HAVE YOU FELT. . .		1= VERY SLIGHTLY OR NOT AT ALL 2= A LITTLE 3=MODERATELY 4= QUITE A BIT 5=EXTREMELY	
1. interested	1 2 3 4 5	11. irritable	1 2 3 4 5
2. distressed	1 2 3 4 5	12. alert	1 2 3 4 5
3. excited	1 2 3 4 5	13. ashamed	1 2 3 4 5
4. upset	1 2 3 4 5	14. inspired	1 2 3 4 5
5. strong	1 2 3 4 5	15. nervous	1 2 3 4 5
6. guilty	1 2 3 4 5	16. determined	1 2 3 4 5
7. scared	1 2 3 4 5	17. attentive	1 2 3 4 5
8. hostile	1 2 3 4 5	18. jittery	1 2 3 4 5
9. enthusiastic	1 2 3 4 5	19. active	1 2 3 4 5
10. proud	1 2 3 4 5	20. afraid	1 2 3 4 5

Adapted from Watson, D., Clark, A. L., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*, 54, 1063-1070.

Scoring: Positive affect scores are determined by summing items 1, 3, 5, 9, 10, 12, 14, 16, 17, and 19. Negative affect scores are determined by summing items 2, 4, 6, 7, 8, 11, 13, 15, 18, and 20.

APPENDIX D: The Emotion Amplification and Reduction Scales

Instructions: <i>Please respond to each of the following statements as they apply to you and your experience with your feelings and emotions. Use the following response scale:</i>	1 = Not at all true for me 2 = Somewhat true for me 3 = Moderately true for me 4 = Very true for me			
1. I can use my emotions or feelings to my advantage.	1	2	3	4
2. I can control my emotional reaction to events or situations.	1	2	3	4
3. When the need arises, I can cut short an emotional response.	1	2	3	4
4. I can stop an emotion before it overwhelms me.	1	2	3	4
5. Prior to a stressful situation, I can get myself into a calm state that actually prevents me from feeling bad when the stressful event happens.	1	2	3	4
6. If I want to, I can get myself emotionally “charged up”.	1	2	3	4
7. I can get emotionally “revved up” to enhance my performance.	1	2	3	4
8. If I wanted to, I could turn UP the intensity level of whatever emotion I may be feeling.	1	2	3	4
9. I can do things that will enrich my emotional experience.	1	2	3	4
10. When I know in advance that I will be faced with an exciting or stressful situation, I could (if I wanted to) remain calm.	1	2	3	4
11. I can choose to remain calm in almost any situation.	1	2	3	4
12. I can readily make myself tone down the intensity of any emotion that I might be feeling.	1	2	3	4
13. I can deepen the feeling of an existing emotion.	1	2	3	4
14. When I know in advance that an upcoming situation is going to make me feel a particular emotion (such as sadness or anger), I am able to do things that prevent the feelings from occurring when that situation arises.	1	2	3	4
15. I can do things that will deepen my emotional experience.	1	2	3	4
16. I can harness the energy of my emotions to enhance my performance.	1	2	3	4
17. No matter how intensely I may be feeling a particular emotion, I can almost always make myself calm down.	1	2	3	4
18. I can hold on to a feeling or emotion.	1	2	3	4

From Hamilton, N., Karoly, P., Gallagher, M., Stevens, N., Karlson, C., & McCurdy, D. (2009). The assessment of emotion regulation in cognitive context: the emotion amplification and reduction scales. *Cognitive Therapy and Research*, 33, 255-263.

Scoring: Amplification scores are determined by summing items 1, 6, 7, 8, 9, 13, 15, 16, and 18. Reduction scores are determined by summing items 2, 3, 4, 5, 19, 11, 12, 14, and 17.

APPENDIX E: Informed Statement of Consent

INTRODUCTION

The Department of Psychology at the University of Kansas supports the practice of protection for human subjects participating in research. The following information is provided for you to decide whether you wish to participate in the present study. You may refuse to participate in this study. You should be aware that even if you agree to participate, you are free to withdraw at any time. If you do withdraw from this study, it will not affect your relationship with Research Medical Center's VITA Eating Disorders Program, the services it may provide to you, or the University of Kansas. In fact, staff from Research Medical Center's VITA Eating Disorders Program will not be informed about participation or non-participation in this study.

PURPOSE OF THE STUDY

The purpose of this study is to examine the role that emotions and emotion regulation play in the vulnerability to and the maintenance of disordered eating behavior.

PROCEDURES

You will be asked to answer questions and complete a set of questionnaires, which will include questions about eating behavior, eating attitudes, and emotions/affect. This will take approximately 30 minutes to complete. The questionnaires will be used for research purposes only and stored in a secure location.

If you decide to participate, please seal the completed questionnaire and one statement of informed consent in the envelope. Return the envelope to the intake nurse. Please keep one of the informed consent statements for yourself. If you decide not to participate, seal the blank forms in the envelope and return them to the intake nurse.

RISKS

There are no foreseeable risks to participating in this study.

BENEFITS

It is unlikely that there would be any direct benefits to you. This study may increase our understanding of the role that emotions and emotion regulation play in disordered eating attitudes and behavior.

PAYMENT TO PARTICIPANTS

You will not be paid for your participation in this study.

PARTICIPANT CONFIDENTIALITY

The information you provide will be ANONYMOUS and we will have no way to link the answers you provide to your name or identifying information. The researchers will use a participant number.

Permission granted on this date to use and disclose your information remains in effect indefinitely. By reading this statement and completing the questionnaires you give permission for the use and disclosure of your information for purposes of this study at any time in the future.

INFORMATION TO BE COLLECTED

To perform this study, researchers will collect information about you. This information will be obtained via questionnaires. The questionnaires will assess demographic information, eating behavior, eating attitudes, and emotions/affect, and your body mass index.

The information will be kept confidential. Your name cannot be associated in any way with the information collected about you or with the research findings from this study. The researchers will use a participant number instead of your name.

The information collected about you will be used by the principal investigator, Danyale McCurdy, and members of the research team. Your data will be ANONYMOUS. We will have no way to connect the information you report to you.

Permission granted on this date to use and disclose your information remains in effect indefinitely. By reading this statement and completing the questionnaires you give permission for the use and disclosure of your information for purposes of this study at any time in the future.

CANCELLING THIS CONSENT AND AUTHORIZATION

You may withdraw your consent to participate in this study at any time. You also have the right to cancel your permission to use and disclose information collected about you, in writing, at any time, by sending your written request to: Danyale McCurdy, 323 Fraser Hall, University of Kansas, 1415 Jayhawk Blvd., Lawrence, KS 66045-7556. If you cancel permission to use your information, the researchers will stop collecting additional information about you. However, the research team may use and disclose information that was gathered before they received your cancellation, as described above.

QUESTIONS ABOUT PARTICIPATION

Questions about procedures should be directed to the researcher(s) listed at the end of this consent form.

PARTICIPANT CERTIFICATION:

I have read this Consent and Authorization form. I have had the opportunity to ask, and I have received answers to, any questions I had regarding the study. I understand that if I have any additional questions about my rights as a research participant, I may call 785-864-7429 or 785-864-7385 or write the Human Subjects Committee

Lawrence Campus (HSCL), University of Kansas, 2385 Irving Hill Road, Lawrence, Kansas 66045-7563, email dhann@ku.edu or mdenning@ku.edu.

By returning this questionnaire, I agree to take part in this study as a research participant. In addition, I affirm that I am at least 18 years old, and that I have received a copy of this Consent and Authorization form.

Researcher Contact Information:

Danyale McCurdy, M.A.
Principal Investigator
Department of Psychology
323 Fraser Hall
University of Kansas
Lawrence, KS 66045
785-864-4121
e-mail: danyale79@msn.com

Nancy Hamilton, Ph.D.
Faculty Supervisor
Department of Psychology
446 Fraser Hall
University of Kansas
Lawrence, KS 66045
785-864-9827
nancyh@ku.edu

APPENDIX F: Recruitment Letter and Instructions

Dear Potential Participant:

Thank you again for your time. We appreciate that you are taking time to fill out this questionnaire.

All of the information contained in this questionnaire is **STRICTLY AND COMPLETELY ANONYMOUS** and no one will know whether you choose to participate in this study or not.

If you **DO** wish to participate:

Seal the completed questionnaire and **ONE** of the statements of informed consent inside the envelope and return the envelope to the intake nurse.

If you **DO NOT** wish to participate:

Seal the envelope and return blank questionnaires to the intake nurse.

General Instructions

1. Please read carefully the instructions for each questionnaire. **PAY SPECIAL ATTENTION TO THE TIME FRAMES LISTED IN SOME OF THE INSTRUCTION SETS.**
2. Although there is no penalty for skipping a question, **PLEASE DO YOUR BEST TO ANSWER ALL OF THE QUESTIONS.**
3. Please choose **ONE VALUE FOR EACH QUESTION.** Sometimes you may feel that your true answer is somewhere in between the values provided. If that is the case, please **CHOOSE THE CLOSEST OR MOST CORRECT ANSWER.**
4. **THERE ARE ABSOLUTELY NO RIGHT OR WRONG ANSWERS TO ANY OF THE QUESTIONS.**

APPENDIX G: Demographic Questionnaire

1. Your age: ____ years

2. Are you currently a full time high-school student: YES NO

2a. If so, are you classified as a . . .

Freshman	Junior
Sophomore	Senior

3. Are you currently a full time university student: YES NO

3a. If so, are you classified as a . . .

Freshman	Junior
Sophomore	Senior

3b. What is your major _____

4. Total years of Education (so far):

<input type="checkbox"/> 0-11 years (High school incomplete)	<input type="checkbox"/> High school completed
<input type="checkbox"/> Post high-school, business or trade school	<input type="checkbox"/> Associates Degree (AA)
<input type="checkbox"/> Bachelor's Degree (BA, BS)	<input type="checkbox"/> Master's Degree (MA)
<input type="checkbox"/> Doctoral Degree (Ph.D.)	<input type="checkbox"/> Physician (MD)

5. Employment Status.

☐ Work full-time outside the home
☐ Work part-time outside the home
☐ Unemployed-looking for work
☐ Unemployed-stay at home parent

6. What ethnic or racial group do you identify with? Check all that apply.

_____ White (Caucasian) _____ Black (African American)
 _____ Asian/Pacific Islander _____ Hispanic (Latino/a) _____
 _____ Indian/Native American: tribal affiliation _____
 _____ Other (Specify) _____

7. At home, do you speak a language in addition to, or other than English

_____ No (0)
_____ Yes (1)

8. Annual Family Income

_____ 1. Under 10,000	_____ 2. 10,000 to 19,999	_____ 3. 20,000 to 29,999
_____ 4. 30,000 to 39,999	_____ 5. 40,000 to 49,999	_____ 6. 50,000 to 59,999
_____ 7. 60,000 to 69,999	_____ 8. 70,000 to 79,999	_____ 9. 80,000 to 89,999
_____ 10. 90,000 to 99,999	_____ 11. over 100,000	

APPENDIX H: Power Analysis

Power Analysis: Alpha = .05

N	Effect Size				
	0.40	0.50	0.60	0.70	0.80
10.00	0.45	0.64	0.81	0.91	0.97
11.00	0.49	0.69	0.85	0.94	0.98
12.00	0.53	0.74	0.88	0.96	0.99
13.00	0.57	0.77	0.91	0.97	0.99
14.00	0.61	0.81	0.93	0.98	
15.00	0.64	0.84	0.95	0.99	
16.00	0.67	0.86	0.96	0.99	
17.00	0.70	0.89	0.97	0.99	
18.00	0.73	0.90	0.98		
19.00	0.76	0.92	0.99		
20.00	0.78	0.93	0.99		
21.00	0.80	0.95	0.99		
22.00	0.82	0.96	0.99		
23.00	0.84	0.96			
24.00	0.86	0.97			
25.00	0.87	0.98			
26.00	0.89	0.98			
27.00	0.90	0.98			
28.00	0.91	0.99			
29.00	0.92	0.99			
30.00	0.93	0.99			
31.00	0.94	0.99			
32.00	0.94	0.99			
33.00	0.95				
34.00	0.96				
35.00	0.96				
36.00	0.97				
37.00	0.97				
38.00	0.97				
39.00	0.98				
40.00	0.98				
42.00	0.99				
44.00	0.99				
46.00	0.99				
48.00	0.99				
50.00	0.99				

Conclusion: In order to find a large effect size (conventionally .40) with a sufficient power (conventionally .80), 21 participants in each group are needed.